THE JOURNAL OF PERIODONTOLOGY

January
1937

Published by
THE AMERICAN ACADEMY OF PERIODONTOLOGY
VOLUME 8.
NUMBER 1.

CONTENTS

		Page
Systemic Diseases to be Considered in Paradentosis	SAXTON T. POPE, JR.	7
The Influence of a Local Excess of Tri-Calcium Phosphate in Controlling Hemorrhage and Healing of Bone Following Injury	J. A. SINCLAIR O. C. BARKER	13
Results of Experimental Root Canal Therapy	C. MERLE DIXON U. G. RICKERT	19
A Case of Reattachment?	DICKSON G. BELL	30
Radiology in Prevention, Diagnosis and Treatment of Periodontoclasia	GEORGE R. WARNER	33
Nutrition and Malnutrition from the Laboratory and Clinical Aspects	JOHN ALBERT MARSHALL	39
The William J. Gies Testimonial	ARTHUR H. MERRITT	45
The Prevention of Periodontoclasia	HAIDEE WEEKS	47
Incidents of Office Practice—Method of Taking Case History	FREDERICK A. BRICKER	52
A Stubborn Case of Vincent's Infection	JOHN H. MACKAY	53
The Treatment of Extensive Periodontoclasia of the Lower Anterior Teeth	R. DEAN CREAMER	53
Indication and Contraindication for Subgingival Curettage in the Treatment of Paradentosis	DICKSON G. BELL	55
The Price of Professional Progress		58
Committee Reports		60
Book Reviews		62

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THE JOURNAL OF PERIODONTOLOGY is published to serve as the professional journal of periodontia; to keep dentists in touch with research being carried on in this field and related sciences; to stimulate greater interest in the supporting tissues of the teeth and their relation to oral and general health. The plan to continue the publication of abstracts indefinitely will make the Journal a fund of extensive information, readily available to students, teachers, researchers and clinicians.

The Journal is the official organ of the American Academy of Periodontology and will contain therefore those papers of scientific interest which are presented at the annual meetings of that society. The proceedings of the 1936 annual meeting are published in this number. The July, 1937 issue will contain the abstracts of articles on periodontia which appeared during the year of 1936.

THE Twenty-fourth Annual Meeting of the American Academy of Periodontology will be held in Atlantic City, N. J., July 8, 9 and 10, 1937, at the Claridge Hotel. Members of the American Dental Association are urged to attend.

THE JOURNAL OF PERIODONTOLOGY

JANUARY, NINETEEN HUNDRED THIRTY-SEVEN

Systemic Diseases to be Considered in Paradentosis*

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THE relation of paradentosis to constitutional disease has been suspected for some time. During recent years, the subject has been attracting increasing attention. At present, it does not seem possible to define the relation. It may be coincidental, contributory, causal.

Most of the systemic disorders belong to three groups. These are intoxications, dietary deficiencies, and defects of internal glandular control. The dietary deficiencies are those of mineral intake—and of the accessory food factors, called vitamins.

Phrase it another way. The disorders under consideration seem to be those affecting the mobilization and storage of minerals which form bone within the human body. These minerals are known to include calcium, phosphorus, magnesium, iron, and copper. One is inclined to mention calcium alone, to the exclusion of the other fractions. For one thing, calcium has been the subject of the greatest bulk of experimental work. Further, it is inconvenient to name the entire list each time one designates the bone minerals . . . The future remarks of this paper, therefore, are apt to slight bone constituents other than calcium. The oversight is to be construed as a nominal one, only.

Let one attempt to review the status of the constitutional factor in paradentosis. He is made quickly to realize the strong diversity of present opinion. The abstracts of current dental literature as presented by the Journal of this Society (the Journal of Periodontology) are ample indication of the situation.

The speaker wishes to isolate, for examination, five elements which he regards as contributory to existent confusion.

(1) Information about certain phases of calcium metabolism is imperfect.

(2) Standards for diagnosis of several of the nutritional and glandular disorders involved are imperfect.

(3) Biometrical analysis on a representative sample appears to be lacking. Routine findings by standard physical and laboratory methods have not been amassed.

*Read before the American Academy of Periodontology, July 9, 1936, San Francisco, Calif.

(4) Unwarrantable finality and a tendency to preconception are apparent in the existing literature.

(5) It appears that classifications of paradentosis are incomplete, and it seems doubtful that diagnostic standards suffice to exclude all closely allied disorders.

(1) Imperfect information about some phases of calcium metabolism.

Even the technical procedures involved offer recurrent discouragement to workers in the field of mineral metabolism. One acknowledges that the same criticism might be made of any branch of medical research. Still, with calcium the limits of exact information are immediate. Reliable results are particularly hard to derive. The name of Joseph C. Aub dominates the subject. To him we are indebted for the study of calcium balance in the human.

The method may be sketched as follows: First, three blood serum determinations are made. These are calcium, phosphorus, and phosphatase. Phosphatase is an enzyme, hydrolysing phosphoric esters. It probably favors deposition of calcium phosphate in the bones.

Next, the patient is presented with a rather unattractive menu. Totally invariable, the same items are offered for each meal, each day. Amounts are weighed carefully. Superficially it looks like chicken breasts, ginger ale, honey and calcium-free fudge. More profoundly, it turns out to be a relatively calcium-poor, neutral ash diet computed to the fraction of a gram . . . A capsule of carmine dye is given along with the first serving.

The appearance of this colorful marker in the stool is the signal for a decorus but intense activity on the part of the nursing staff. Nothing unusual is required of the patient. He must continue to eat his food—and dispose of it normally. He must tell the truth about any morsel which he refuses.

Meanwhile, all stools and urine voidings are collected into special containers. These have been washed calcium free.

The process continues for a minimum of three days. A second carmine capsule is ingested. Its appearance in the stool marks the termination of the dietary period. A second blood sample for calcium, phosphorus, and phosphatase is collected. The patient is liberated. The nursing staff relaxes. It congratulates itself, if it has scored a perfect record for stools and urine samples into proper containers.

The excreta are analysed for mineral output . . . Statements of ingested totals come from the diet kitchen. The two are compared. Under favorable circumstances, one has the result—a normal, a positive, or a negative calcium balance. The positive balance implies that a greater amount has been ingested than can be reckoned from the dejecta. The negative balance implies the reverse.

In the case of a significantly negative or positive balance, one is left to inter-

pret how it has come about. Sometimes the etiologic factor stands clearly demonstrated. Frequently, however, one is brought back to grips with the multiple and inter-related dietary and internal glandular factors controlling mineral metabolism.

(2) Imperfect standards for the diagnosis of nutritional and glandular factors.

The following morbid (or physiologically doubtful) states have been suspected of influencing the paradentium, and must be included in any representative partial list.

1. Pregnancy and lactation.
2. Hyperthyroidism.
3. Hypothyroidism.
4. Hyperpituitarism (eosinophilic, or acromegaly).
5. Basophilic pituitary adenomata (Cushing's entity).
6. Adrenal cortical tumors.
7. Hyperparathyroidism (von Recklinghausen's disease).
8. Diabetes mellitus.
9. Osteitis deformans (Paget's: of doubtful parathyroid affiliations).
10. Vitamin C deficiency.
11. Vitamin D deficiency.
12. Acid ash diet.
13. Toxic factors, prominently lead poisoning.

It is not expected that the entire list of disorders will be familiar to all of my audience. Adrenal cortical tumors, acromegaly, basophilic adenomata, Paget's and Von Recklinghausen's diseases form a highly spectacular group. Four of the five are proven endocrine dysfunctions. The same four are locally benign neoplasms. The fifth is *suspected* to be related to endocrine dysfunction. All five are marked by gross skeletal changes. All five, as fullblown clinical pictures would qualify the sufferer for exhibition in a circus side-show—and frequently do. It is their advanced skeletal changes which make them pertinent to our discussion.

There is an obvious method for testing the relation of paradentosis to these disorders. One might establish a sizeable universe of each, and compare its incidence of paradentosis with that in a normal or control group.

I am told that this has been done, in the case of diabetes, by Hirschfeld. One would think it must have been, also, in pregnancy and lactation. It is lactation, incidentally, which is considered the greater drain. Which tends to make one think of cows . . . I do not know that they have been the subject of any detailed research.

The method might be applied most easily to gross hypothyroidism in endemic areas such as Switzerland, (or might have been until recently) . . . It ought

also to be applicable to clinical hyperthyroidism . . . Equally so to a rachitic group. One thinks of the mouths of the poor in Glasgow. An immediate objection, however, becomes apparent. Their "porridges," their lamb, tea and crumpets must constitute an acid ash diet . . . Further, we cannot ignore the fact that rachitis has exerted its influence on the mouth from birth. The search for constitutional paradental factors suggests that one look for adult influences on fairly formed teeth.

It might be a little more difficult to assemble an acromegalic group but ought still to be possible.

Basophilic adenomata, adrenal cortical tumors, hyperparathyroidism, osteitis deformans are to be regarded as frank medical rarities. Proven basophilic adenomata still number fewer than fifty cases . . . Studies of these clinical states, therefore, must be governed by occurrence. Oddly, frank adult scurvy is also to be listed as a medical rarity in this country.

There is an added complication of which the full weight has been felt only recently. Certain of the listed disorders can—and frequently do—exist in mitigated subclinical forms. I refer to the dietary deficiencies and hypothyroidism, especially.

To particularize: The fullblown picture of adult hypothyroidism or myxoedema is familiar to everyone . . . Classically, the patient is heavy, dry of skin. He has thick ankles and a thick tongue. The eyelids are puffed. Little pads of fat lodge above the clavicles. Hair coarsens and disappears from the latter half of the eyebrows and from the forehead at the temples. The cheeks are bright like polished apples. Speech and wit are slow. In women, the interval between menstrual periods lengthens. The heart rate is slow; often sixty beats or less per minute. Basal metabolic rate determinations range between minus 25 and minus 40.

About four years ago it was proposed by a group of physicians at the Harvard University to treat advanced heart disease by removal of the thyroid gland. They felt that artificial hypothyroidism, by lowering the rate of blood flow, might influence such a group favorably. A considerable number of patients was operated. The thyroid gland was removed, totally. The results of this series have been debatable from the cardiac standpoint. The series, however, has given an opportunity to study more closely than ever before, the levels at which the various symptoms and signs of the hypothyroid state develope.

General observation has long placed the zone of normal basal metabolic rate between plus and minus 10 per cent. Total absence of thyroid tissue gives minus 40 or a little more. The results of the Harvard cases demonstrated that symptoms tend to become evident between minus 10 and 20 per cent. Symptoms are defined as the subjective aspects of disease. For hypothyroidism, they are fatigue, inertia, lassitude. Physical *signs* of the condition become obvious only between minus 20 (or a little more) and minus 30.

It is to be acknowledged, therefore, that there exists a zone of definite but physically inobvious hypothyroidism.

Since such a zone exists without physical signs, on what can one rest one's diagnosis? To the best of the speaker's knowledge, it can be rested only on one—(or several)—of four things.

1. A consistent basal metabolic rate between the stated limits.

2. A blood cholesterol determination considerably in excess of the accepted limits of normal—safely over 200 mg. per cent.

3. The voluntary testimony of the patient to a debility which no physical finding satisfactorily explains.

4. Correction of most of the above by a properly managed dose of thyroid extract.

Therapeutically, the thyroid is the most easily manageable of the glands of internal secretion. Its hazards are real and have been universally stressed. Stressed, possibly to the obfuscation of a general realization that its successful management gives brilliant results.

It is not the purpose of this paper to indicate any conclusions emanating from the Department of Dental Medicine at the University of California. During the past two years a considerable number of patients has passed through the department's hands. Routinely, each patient has been submitted to the diagnostic approach which has been outlined by Dr. Becks.

The paradentosis group has not yet been analysed. It has, however, shown a surprising tendency toward minus basal metabolic rates. Very few of the patients have exhibited physical stigmata of hypothyroidism.

The trend is definite enough to have engendered suspicion in the minds of all concerned. Basal rate technique has been scrutinized. The machines have been tested against control subjects. Nothing has upset the observation. The series contains a surprising number of persons without physical signs now considered as orthodox, but having consistently low metabolic rates.

(3) Lack of any group of sufficient size for statistical study.

Ultimately it is hoped to submit the group to biometrical study. This is the method suggested above, in application to gross endocrine dysfunction. The method is suitable both to a disease entity and a single sign. The correlation may be determined between paradentosis and inobvious low metabolic rate, for instance. And, since biometry is to research almost what the auditor is to business, the results of the sifting will be watched with keenest interest.

(4) An unwarrantable tendency in the existing literature.

The speaker has proposed that finality and preconception are apparent in the existent literature . . . He would like, if he may, to submit this possibility to his audience, without comment.

(5) . . . Present classifications of paradentosis to not exclude allied disorders.

The proposition that diagnostic standards for paradentosis fail to exclude allied disorders, is made with some timidity.

While my information on this subject is fragmentary, I am instructed that unanimity of opinion does not exist. Congruous situations, medically, makes me doubtful that a satisfactory judgment can be passed on the constitutional factor, under terms of present classification.

An outstanding medical analogy would be arthritis. First, it had to be realized uniformly that there were really arthritides. Then the separate entities had to be defined. The chronic form was divided into two great types . . . One called *hypertrophic;* proven to be a matter of weight bearing, joint wear, and calcium deposit—totally without any infective element. A second type was named *atrophic,* occurring in younger, usually slender individuals. A factor of infection was granted for this type. Focal infections were suspected to be etiological. The situation was almost exactly comparable with the question under discussion today. No universal relation of atrophic arthritis to focal infections was demonstrated—and none of focal infections to atrophic arthritis. Just recently, a plausible predisposing factor has been uncovered; subclinical scurvy, or lack of vitamin C.

Close inspection has demonstrated total absence of association between the hypertrophic form and focal infection.

One thing seems fairly obvious. Studies of the relation between constitutional disorder and paradentosis should not be made. ***Rather, studies of the relation between particular consitutional disorders and particular types of paradentosis.***

An important pre-requisite of this study will be one of the effect of glandular and dietary disorders on basic human histologic tissues—ectoderm, mesoderm, and entoderm.

One must re-emphasize the importance of the biometrical approach. Dependable data, collected in a manner to be serviceable, have not been reported. Valid conclusions on even a small portion of the field will require several years.

Meanwhile, one's attitude on the problem will be determined by the present evidence which has attracted one's attention. Under the circumstances, a personal statement of faith may be admissable.

The speaker conceives constitution as the background.

In particular, his attention is focused on calcium-abstractive factors. Certain of these have been listed above. Certain are granted to exist in physically unrecognizable, subclinical forms.

It may even prove that dental roentgenograms will form a valuable aid to the diagnosis of these states.

Calcium loss and the other intimate histological changes still to be defined—these probably constitute the predisposition, or diathesis.

The immediate, exciting factors, however, are apt to be found locally within the mouth; trauma, infection or both.

The Influence of a Local Excess of Tri-Calcium Phosphate in Controlling Hemorrhage and Healing of Bone Following Injury*

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ACCORDING to recent advances in physiological chemistry made by Leriche and Pollicard of France and Bancroft and Murray of this country, the processes of resorption and ossification of bone following injury are regarded as purely chemical changes taking place extra-cellularly in the tissues around an injury. This modern interpretation, we believe, is applicable to the continuous resorptive process involved in producing the pyorrhea lesion.

The classical description by Clay Ray Murray of the healing of a fracture[1] exemplifies these new beliefs. Murray states:

All fractures heal as do wounds elsewhere, unless there is a mechanical, chemical, or anatomic bar to the healing. This healing, in common with that of wounds of the soft parts, takes place through the medium of new connective tissue known as granulation tissue.

Variations in the character and amount of the healing process in bone, following fracture, are dependent on factors limited to the region of the body involved quite independent of the subject involved. Slow union and nonunion are not influenced by the age *per se* of the patient, by his general state of health, or by the presence of chronic general disease, such as syphilis or cardio-vascular or renal disease. The preponderance of clinical and experimental evidence on the calcium and phosphorus levels of the blood during the healing of a fracture is unquestionably to the effect that there is no correlation between variations in these levels and variations in the healing process. In keeping with this are the clinical findings in osteomalacia and rachitis, in which healing is normal or slightly more rapid than normal after fracture, although there exists an actual negative balance in the former and an acknowledged metabolic disturbance in the latter. The only true nonunions—that is to say, failures of healing—are those due to the mechanical, chemical, physical and anatomic cause.

The role played by soft parts, including periosteal flaps, whether living or dead, and by large fragments of dense bone, if dead, as mechanical bars when interposed between the ends of the bone requires no discussion.

Because of the exceedingly minimal or even total lack of circulatory status, certain regions of the body, even under apparently optimum conditions, are characterized by a slow average healing time and by frequent occurrence of delayed union and non-union in adults. The neck of the femur (subcapital or midcervical), the junction of the lower and middle thirds of the tibia, the scaphoid of the carpus (proximal half), the base of the fifth metatarsal and the junction of the lower and middle thirds of the humerus are characteristic regions.

The influence of infection in producing continuous necrosis by chemical means, thus preventing primary healing, is obvious, and it has been demonstrated that with many suppurative processes there is established, even in the absence of excessive necrosis, an acid pH resulting in the decalcification of bone and in little or no calcium deposition in the granulation tissue, which may, however, be over-abundant."

Concerning the normal healing of bone following injury, Murray says:

On the basis of recent observations, the process in healing of a fracture is one in which, following the injury and the resultant inflammatory processes, an acid pH of the local tissue fluids, dependent on the interrelationship of tissue necrosis and circulatory efficiency, is associated with the de-calcification of the dead bone at the site of the fracture.

* Read before the American Academy of Periodontology, San Francisco, California, July 10, 1936.

Such liberated calcium is held locally by a chemical absorption affinity between it and the fibrin of the hematoma and the collagen of the newly formed connective tissue, i. e., granulation tissue—growing from all directions along the fibrin network at the site of the injury, producing concentration of calcium about the fracture. With increased cell metabolism, and with interrelated increasing efficiency of circulation and the carrying away of the products of necrosis, there is a reversion of this acid pH to the alkaline side, and coincident with this change there occurs a deposition of the locally absorbed calcium in the newly formed connective tissue to form new bone.

Regarding the source of local calcium, Murray says:

Not only does the clinical evidence support the view that the effective source of the calcium used for callus in the primary healing process following fracture is the traumatized bone at the site of the fracture, but experimental evidence bears out the clinical findings and, in the main, adequately supports the view that the levels for blood calcium and phosphorus and the general calcium metabolism have no appreciable bearing on the question of primary healing of fractures and that as a source of calcium the dead bone at the site of the fracture can actually be substituted for by calcium salts of various types introduced in replacement of such dead bone.

According to Haldeman and Moore[3] many workers have injected various salts of calcium and phosphorus at the site of experimental fractures in the hope of influencing the rate of healing. Albee and Morrison[3] injected a suspension of tricalcium phosphate in a gap produced in the radius of a rabbit. The other radius, with a similar defect, served as a control. The average length of time required for union of the fracture treated with calcium phosphate was thirty-one days, as compared with the control, which required an average of forty-two days.

Cretin[4] found that the injection of the various mineral elements of bone between the ends of experimentally produced fracture resulted in a marked acceleration of the formation of callus and bone.

The experiments of Rollo[5] indicate that the injection of calcium glycerophosphate into the tissues around a fracture in rabbits serves to stimulate the formation of callus and bone.

Haldeman and Moore, in their experiment, used three preparations of calcium phosphate (tricalcium, dicalcium, and monocalcium) and calcium glycerophosphate in operative defects, between 0.5 and 1 cm. in length, made in the radii of seventeen rabbits. One side served as a control, while the defect in the opposite radius was filled with a dry, powdered salt of calcium and phosphorus. In using monocalcium phosphate, rabbit No. 20 died in twenty-three days. The defect in the leg which was filled with the calcium developed an abscess and, of course, no healing occurred. In others of the monocalcium series, the control and treated defects healed in the same time.

In the series in which tricalcium phosphate (Fig. 3—experiment 72) was used, the control and the treated defect healed in the same time. In Fig. 5,—Experiment 76, in which the same procedure was followed, there was complete closure of the defect in which tricalcium had been used, while typical nonunion occurred in the opposite radius.

In Fig. 7, Experiment 78, in which dicalcium phosphate was used, both radii closed in approximately the same time.

In Fig. 9, Experiment 82, in which calcium glycerophosphate was used the rate of healing seemed to favor the radius in which no calcium was used.

Haldeman and Moore, from their experiments, concluded that an excess of calcium salts at the site of fracture, either did not influence the rate of healing or retarded it. In no case did the presence of monocalcium phosphate, dicalcium phosphate, or calcium glycerophosphate have a favorable influence. In delayed healing resulting from unknown causes tricalcium prosphate at the site of fracture, appeared to favor the union. In their summary they state, however, "An excess of the local supply of calcium salts is reported almost unanimously as accelerating the rate of union."

Murray[6] carried out investigations in which he filled a gap of 2.5 cm. in a dog's radius with sterile powdered calcium carbonate and calcium phosphate, which was followed by solid bony union. Regarding this, he says:

If we resect 2.5 cm. of a dog's radius and throw it away, leaving the gap in bone to heal as it will, we get a fibrous union filling the gap between the two bone ends with a little ossification just adjacent to the bone ends where we have killed some little bone by our operative procedure.

If, instead of discarding the resected portion, we remove all periosteum and endosteum and break it up into chips and place them in the gap, we get a solid bony union, although, as we have seen from our sections, these bone chips are dead and are autolysed, at least in part.

If, to make sure of their death, we boil the fragments before putting them back, we get bony union notwithstanding.

If we decalcify these fragments before we put them back, we get no evidence of bony union.

Obviously, viability being disposed of as a factor, and everything else except the calcium being disposed of as a factor, the role of these bone chips seems to be to supply calcium for the calcification of the granulation tissue constituting the healing process.

Therefore, I have resected in other dogs the same amount of radius, discarded the fragment and placed in the 2.5 cm. gap powdered sterile $CaCO_3$ and $Ca_2(PO_4)_2$—supplying an artificial source of calcium in the gap previously filled by bone—and solid bony union has resulted.

A number of surgeons have injected various salts of calcium and phosphorus into human beings in the hope of securing an acceleration of bone repair.

Eden[7] injected secondary calcium phosphate at the site of the fracture in three instances of delayed healing in human beings. Consolidation resulted in all of the cases, two of which represented fractures of the femur and one a fracture of the tibia.

In outlining his treatment of a series of six cases in the characteristic delayed or nonunion tibial site, Murray[6A] saucerized the bone in the fracture region in removing the infected bone, the area removed varying from four to seven inches. The wounds treated by packing with vaseline gauze healed by bony union but were deficient in amount leaving a saucer-like depression. In regard to this, Murray states:

Qualitatively we had secured our results, but we had actually removed a tremendous area of bone instead of placing additional bone as grafts.

In others of the series we therefore did what was demonstrated in the cases presented —filled the gap in the tibia created by the saucerization with an artificial calcium source—a combination of powdered calcium triple phosphate and calcium carbonate well mixed with the blood allowed to fill up the cavity. In these cases we not only secured bony union, but filled the tibial defect with bone, at times even above the normal tibial surface level.

The influence of a foreign irritant or infection in producing continuous necrosis by chemical means, resulting in an inflammatory condition in which there is a reversal of the pH of the tissues to the acid side, is the outstanding factor in the resorptive process associated with the pyorrhea lesion. Treatment, therefore, should be directed towards the removal of the irritant and infection, which will be followed by a growth of granulation tissue, and coincident with this there will be return of the pH of the tissues to normal, a medium in which locally absorbed calcium is deposited to complete primary healing.

In the pyorrhea lesion there is no primary healing; the delay in the healing is evidently due to the lack of a local supply of calcium, as there is no dead bone (Kronfeld)[9] at the site of the injury to supply such a source. The successful closures we have had are due, perhaps, to a previous calcium exhaustion as in pregnancy, lactation, or deficient calcium metabolism in which there exists a state of osteo-porosis. Ghormley and Stuck[8] hasten the healing of bone defects in animals by previously feeding a diet deficient in calcium.

A fracture is not healed until "cortical union" takes place; neither is a pyorrhea lesion healed until "cortical closure" is complete. To influence the rate of healing and the replacement of lost bone we have used tricalcium phosphate and calcium carbonate as an artificial source of calcium, as suggested by these investigators. Our results, as to an early "cortical closure," are more than satisfactory; as to the replacement of lost bone we are unable to say as our first cases were disappointing. A changed technique, in which we are able to constantly feed the tissues with calcium and with the addition of calcium carbonate to the triple calcium phosphate, has brought promising results.

In advanced lesions, following operative procedure, tricalcium phosphate is mixed with the blood and the wound closed and sealed, either by Ward's or Kirkland's method, and left intact until granulation tissue has become established in the wound, at which time the seal is removed and powdered calcium carbonate and tricalcium phosphate, in the ratio of 1 to 4, are worked into the granulation tissue with a rubber plunger similar to that used by Vastine[10] and the wound sealed again. This treatment is repeated every two or three days until the granulation tissue has disappeared.

In the less advanced cases, where subgingival currettage is used, powdered calcium carbonate and tricalcium phosphate are worked into the tissues and sealed as described above; this procedure is repeated two or three times a week until the soft tissues are clinically healed.

Interesting observations were made following this procedure; we noted the absence of extreme thermal response and root sensitiveness, the rapidity with which the soft tissues healed, and the almost spontaneous clotting following the mixing of the salts into the blood. When blood is shed, according to Pepper and Farley[11], the blood platelets break up from the lowering of surface tension (coming in contact with a water-wettable surface), the substance liberated by the exploded platelets (thrombokinase, tissue juice, zymoplastic substance, or prothombin) unites with calcium to convert fibrinogen into fibrin. A water-wettable surface smaller than the platelet, such as a bacterial body or colloidal particle, causes the platelet to attach itself to it on contact; the platelet does not explode, but shrinks, and no coagulating substance is liberated.

This clotting is not due to the calcium but to the increased water-wettable surface of the calcium molecule coming in contact with the blood platelets, causing the platelets to explode, liberating a substance which immediately starts coagulation of the blood. The calcium required in the clotting mechanism in amounts necessary for this process is rarely found wanting. Kugelmass[12] in his treatise on "Clinical Control of Chronic Hemorrhagic States in Childhood," states, "Clinically, calcium salts have been used in most hemorrhagic diseases for centuries, with favorable effects but based on erroneous interpretations." Calcium therapy is indicated in abnormal bleeding resulting from vascular dysfunction. Its effect is specific in decreasing capillary permeability. The calcium salts administered have no bearing whatever on elevating the well buffered calcium content of the blood.

A hemorrhagic tendency may follow the loss of the function of contractibility possessed by the capillaries, (Pepper & Farley). In such cases a primary or secondary hemorrhage may occur. In some of the hemorrhagic diseases the influence of calcium will not prevent hemorrhage.

Several months ago I asked a number of dentists to use tricalcium phosphate mixed into the blood clots following operations, and to note any benefits derived from its use in controlling hemorrhage and promoting healing of the soft parts. The following replies are an example of the results:

Dr. W. E. Earley of the Smedley Dental Group, Denver, Colorado, reports thirty-one cases of straight extraction where calcium phosphate was used; bleeding was curtailed, formation of clot good, normal recovery.

Ten cases of impacted third molars where calcium phosphate was used, bleeding curtailed and normal healing in all but two, which were dry sockets, and sedative packs had to be used.

Remarks: Definite curtailment of bleeding, formation of a good clot in majority of cases and apparently quicker healing.

In Dr. Earley's detailed report, case No. 3, "Straight extraction. Patient called during night, reported bleeding freely, pressure pack was used to control bleed-

ing." This case, with two of our own cases, are the only ones reported where hemorrhage followed the use of tricalcium phosphate.

Dr. M. H. Garvin of Winnipeg says:

"We have used considerable of this material (tricalcium phosphate) since receiving it. My partner, Dr. Brown, looks after all the surgery in our office and he has tried the product in a number of ways. He finds that beneficial results are most pronounced when he incorporates the powder in iodoform gauze when a drain is indicated. The wound appears definitely healthier when the drain is removed and the drain itself appears to be in much better shape when removed than formerly. This practice is being followed as a matter of routine. We have not had any severe case of secondary hemorrhage since the material arrived."

Dr. Elmer Best, Minneapolis, writes me the following:

"The calcium phosphate has been received from Merck. It has been tried out extensively in my office. I want to report the most unbelievable results in controlling hemorrhage. I have never seen anything like it and am greatly indebted to you for drawing my attention to it."

SUMMARY

The influence of an irritant, either a foreign body or bacterial, in producing continuous necrosis by chemical means preventing the return of the tissue pH to normal, is the outstanding factor in the production of the nonhealing lesion we call pyorrhea.

Failure of primary healing following treatment is due to a lack of an excess of local calcium at the site of injury. Healing, that is, complete cortical closure, is slow because the healing process depends upon calcium in transit for material to make new bone.

To promote primary healing, and the replacement of at least a portion of lost bone, we suggest the use of tricalcium phosphate and calcium carbonate introduced into the tissues, to provide an excess of the salts at the site of the defect.

To promote healing of the soft parts and control of hemorrhage, tricalcium phosphate is of proven value.

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Results of Experimental Root Canal Therapy*

C. Merle Dixon, D. D. S., and U. G. Rickert, A. M., M. S., D. D. S.
Ann Arbor, Michigan

UNTIL recently the experimental evidence given in support of the practice of root surgery has been largely bacteriologic or roentgenographic. The object of this experiment was to determine whether or not histological studies of necropsy material made from treated and root filled teeth and their surrounding structures of both infected and non-infected "vital" cases would substantiate the bacteriologic and roentgenographic results observed by many investigators.

EXPERIMENTAL ANIMAL

There has been no definite agreement among research workers in the past relative to the laboratory animal which will for experimental purposes most nearly approximate conditions required for human comparisons. Regardless of the favorable comparison of results made by Orban, Stein, Gottleib,[1] there are still those who question the use of the dog. For this type of experiment we have found the dog very satisfactory. The dog is an omniverous mammal and is in many respects biologically similar to man and has many things in common with him. The biological similarity is quite identical as observed by the inflammatory reactions verified by the histological sections.

EXPERIMENT

The bacteriological, roentgenographical and histological results of two types of cases were studied; first, infected cases; second, non-infected cases. The infected case was initiated by inoculating teeth from which the pulps were removed, with a twenty-four hour culture of *Streptococcus viridans* isolated from the root canal of a clinic patient. The non-infected, so-called vital case, comprised teeth from which vital pulps were removed.

In addition to the above, it was planned that the experimentally filled teeth, both infected and non-infected, from one dog should be used for bacteriological studies, and from a second dog the maxilla and mandible were to be removed and histological sections were to be made of the experimentally filled teeth with the attached adjacent tissues. The experiment was to continue for 1½ years after completion of root canal surgery. Two dogs were carried for this length of time while a third died after seven months.

The infected cases were treated after the manifestation of alveolar bone

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*Read before the American Academy of Periodontology at San Francisco, California, July 10, 1936.

*The authors wish to express their appreciation to Faith P. Hadley, M. S., D. P. H., Mary C. Crowley, A. B., M. S. P. H., and Rogene Seymour Wilmot, A. B., M. S. for assistance in the bacteriological part of this paper.

1. Gottlieb, B., Orban, B., and Stein, G.: Das Problem der Wurzelbehandlung—("The Problem of Root Treatment"), Zeitschrift für Stomatologie. 8th Int. Dental Congress, 1931, Sect. 1-5, p. 34.

destruction was perceptible from roentgenograms. From this step on, both types of cases were treated and filled (Kerr Sealer and gutta percha) as like cases are cared for in our clinic.[2]

RESULTS

Bacteriological results: From one experimental animal both the infected and non-infected cases were cultured before necropsy. Cultures were obtained by the window method. This method has been used for years by the late Dr. Chalmers J. Lyons, and Dr. J. W. Kemper of our Oral Surgery Department, and Dr. U. G. Rickert, and has proved very successful. After preparing a sterile field, the incision is made several millimeters above the gingival margin, leaving a thin strip of tissue to which to suture, and the incision is carried in a half moon shape up, over, and somewhat beyond the apex of the tooth to be cultured. The same type of incision is made for an apicoectomy, except somewhat wider, which gives more access to the field of operation. The mucous membrane flap is held back with retractors and the alveolar plate is removed, exposing the apex to be cultured or resected, or both. After obtaining the culture material it was managed essentially the same as those prepared by the Rosenow method.[3]

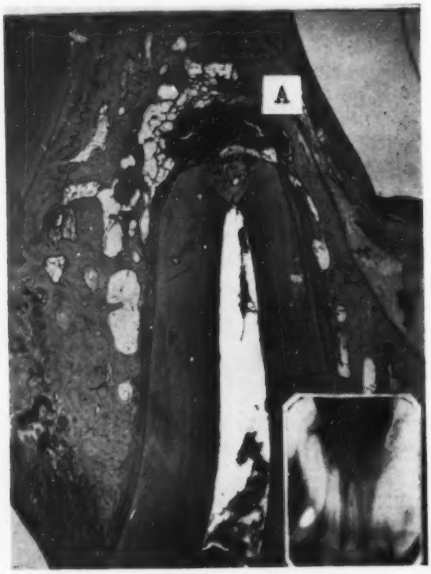

FIG. 1—Case No. 3-E. Upper left canine showing a chronic exudative apical parodontitis. This case was not treated but remained as a source of essential infection throughout experiment. Insert. Roentgenograph showing diffuse rarefying osteitis.

Control cultures were made by scraping the area about the apex and the inoculum transferred to suitable culture media. (.2% glucose, .1% agar, ascitic fluid and beef infusion broth.) This semi-solid media is used to culture teeth in the University of Michigan School of Dentistry and has proved very satisfactory for growing most types of organisms isolated in these cases.

Cultures made by the Rosenow method after five days incubation showed spore formers. Control cultures in semi-solid media showed no growth after incubation of seven days except one which was a spore former of the usual air contaminant type.

Roentgenographical Results: After 1½ years following completion of root canal treatment, we can interpret, in some instances, new bone formation about the apices of the infected teeth. The non-infected cases present a normal picture of the parodontal structures throughout the experiment.

2. Dental Cosmos, March, 1928.
3. J. Dental Research, Vol. 1, p. 205, 1919.

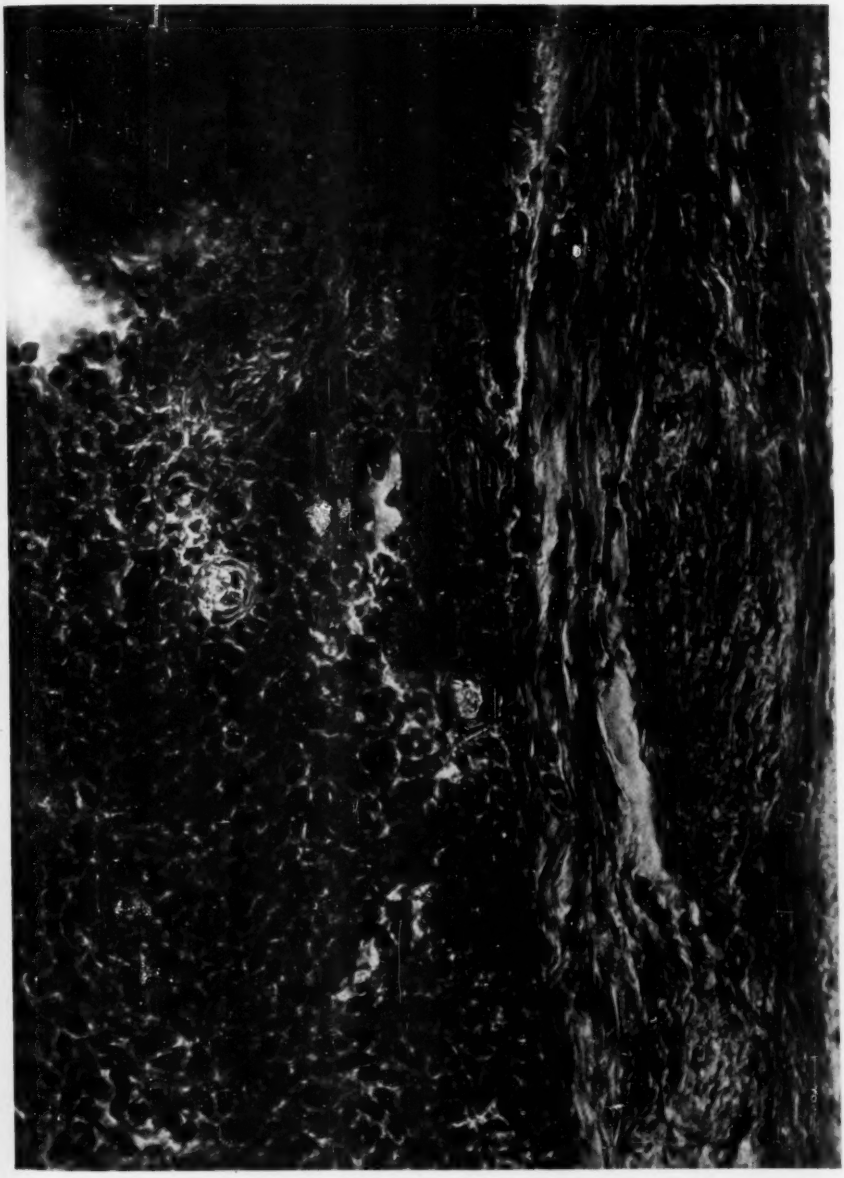

FIG. 2—Higher magnification of Figure 1 area (A) showing fibroblastic proliferation at the right; lymphocytes, plasma cells and walled blood vessels on the left. Leukocytes are so few none can be seen in this field showing a transition from an active purulent to a chronic productive apical parodontitis.

Histological Results: It was originally planned to leave two infected teeth as a source of essential infection in the experimental animal, therefore, the upper right and left canines were not treated. Microscopically, they showed a chronic exudative apical parodontitis. Fig. 1 and 2—Case No. 3-E. It is through the interpretation of this reaction that we believe the use of the dog in this experiment to be justified. To explain this further, we see in this inflammatory process a transition from an active purulent process to that of a chronic exudative and proliferative process evidenced by a marked increase in lymphocytes and plasma cells and only an occasional remaining polymorphonuclear leucocyte. This transition of the purulent inflammation, as seen in the dog, is identical to purulent inflammations in the mouths of human beings, as for instance, acute purulent parodontitis—pyorrhea, gingivitis. If the acute condition is not treated or is unsuccessfully treated and subsequent attacks occur, a gradual transition of the

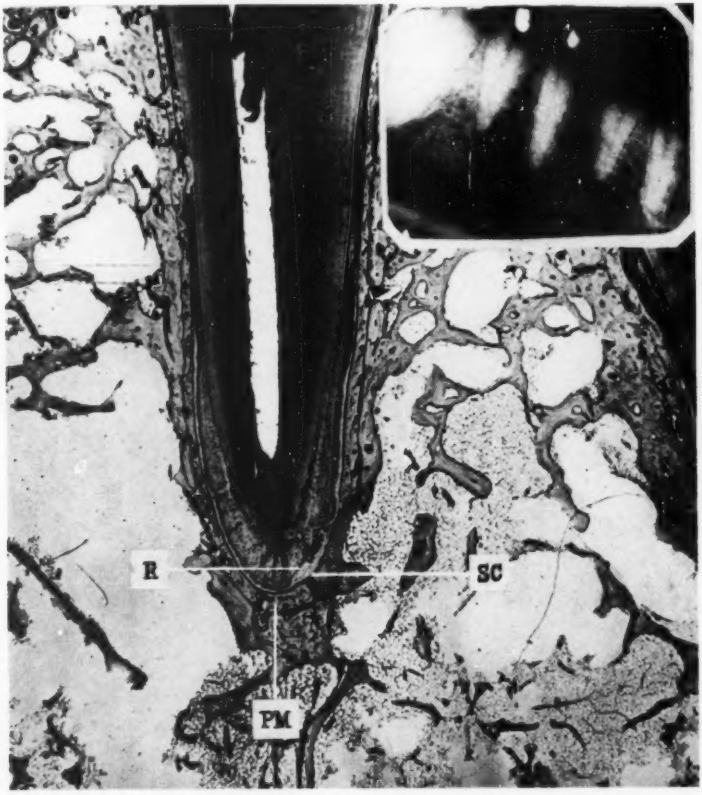

FIG. 3—Case No. 36-E. Infected Case. Distal root of lower left molar, one year and five months after root canal therapy showing resorption line at R; secondary cementum at (SC); and normal periodontal membrane at (PM). Insert. Roentgenograph rarefying osteitis 3½ months after inoculation.

exudate is seen, i. e., from a purulent type to that of an exudate composed of many plasma cells, lymphocytes and fibroblastic proliferation—scar tissue formation, the chronic stage of inflammation.

Infected Cases—

The infected cases showed regeneration in kind by the manifestation of secondary cementum deposited over previously resorbed cementum and normal periodontal membrane over the secondary cementum. Fig. 3—Case No. 36-E and Fig. 4—Case No. 9-E. There are, of course, instances in which there was a residual inflammatory process. This is to be expected because it would depend en-

FIG. 4—Case No. 9-E. Infected case. Distal root of lower left molar seven months after root canal therapy showing the obliteration of a lateral foramen (I) by secondary cementum (SC); normal periodontal membrane (PM). Insert. Roentgenograph showing rarefying osteitis four months after inoculation.

tirely on the extent of destruction produced by the induced streptococcus infection, as to the length of time necessary for complete resolution and regeneration. Here we believe additional work must be carried on over a longer period of time to ascertain the end result in such cases. From the 1½ year cases, we have evidence to warrant an opinion of that picture. Fig. 5—Case No. 38-E. This case shows that there was a rather marked reaction at one time. Widespread alveolar bone destruction and also destruction of cementum, but, as we find it now as chronic productive apical parodontitis, it presents every phase of going on to complete resolution. There is no active alveolar bone destruction at this time, but quite to the contrary, as we see a marked fibroblastic proliferation which embryologically is destined to form new alveolar bone, secondary cementum, and regeneration of periodontal membrane.

FIG. 5—Case No. 38-E. Infected case. Lower left anterior, one year, 5 months after root canal filling. A chronic exudative and productive apical parodontitis. This reaction also shows a transition from an active purulent to a chronic productive infiltrate.

The next case (Fig. 6—Case No. 9-E) shows quite a remarkable tolerance toward an excess of filling material. This was the first tooth to be filled, and, since the filling was not adequately adapted to the walls of the canal, it was removed. Either in removing the filling material or refilling the canal, some material was unintentionally pushed beyond the apex. There is scar tissue surrounding an elongated mass of the sealer, quite remote, however, from the apex. Fig. 7—a higher power of Fig. 6—shows a marked fibroblastic proliferation and a tendency of ingrowth between the material, showing it to be quite a bland substance and well tolerated. There is, of course, some inflammation present, but one must remember that this was originally an infected case and some residual inflammation should be expected. Another section of the same specimen shows periodontal membrane growing between previously resorbed cementum and excess Kerr Sealer, and over the outer surface of the same piece of sealer we see a deposit of secondary cementum. This mass in time might be entirely encapsulated with secondary cementum since this much is present after seven months.

There was one infected case (Fig. 8—Case No. 61-E) of particular interest from those used for bacteriological studies, that showed such definite rarefactions about the apices by roentgenographs that we decided to culture the mesial root,

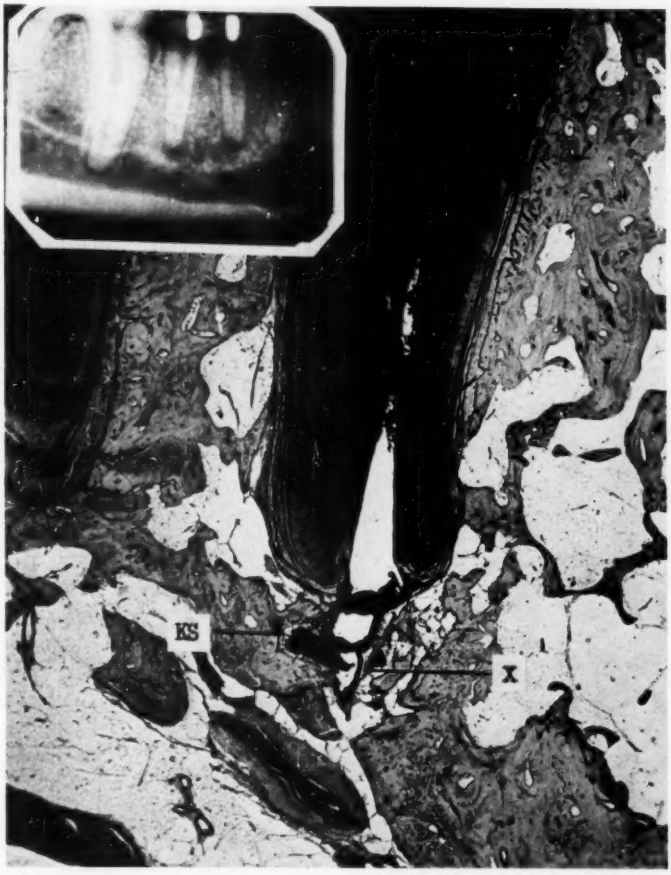

FIG. 6—Case No. 9-E. Infected case. Mesial root of Fig. 4, a lower left molar 7 mo. after root canal therapy (K. S.) Kerr Sealer beyond apex surrounded by scar tissue and fibroblastic proliferation at (X); see high power Fig. 7. Insert. Roentgenograph taken four months after inoculation—rarefying osteitis.

which was slightly over-filled, and retain the distal root for histological study. The mesial root culture was negative. The pathological report of the distal root was a chronic productive inflammation showing newly formed cementum.

Vital Cases—

With but one exception, which showed a mild productive inflammation, there was no destruction of the alveolar plate or the periodontal membrane.

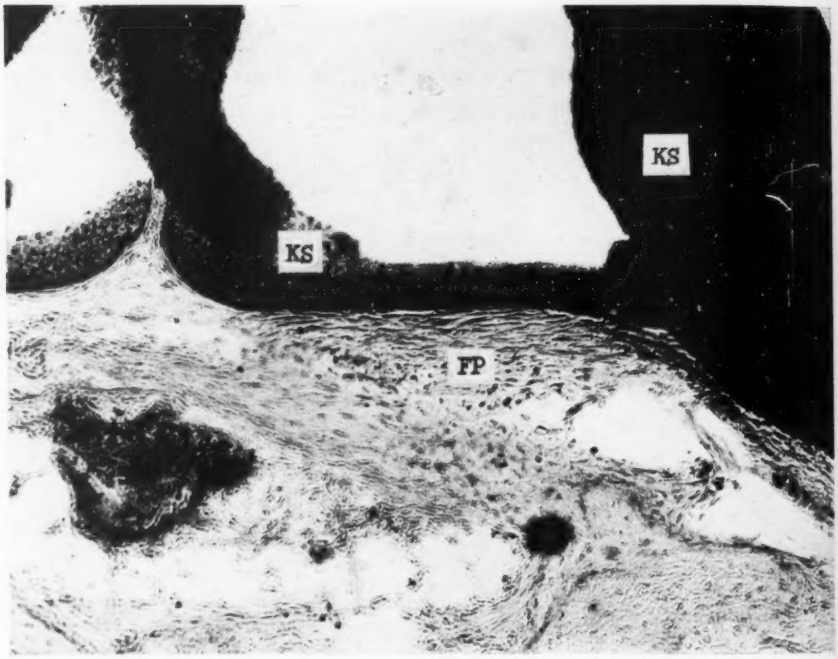

FIG. 7—Infected Case. Higher magnification of Fig. 6 (X). Non-inflammatory fibroblastic proliferation (FP)—adjacent to and growing between excess Kerr Sealer (KS) demonstrates the actual tolerance of this material to the parodontal tissues.

Figure 9—Case No. 13-E shows normal periodontal membrane with its various types of fiber bundles and spaces containing nerves and blood vessels. An accessory canal on the left shows an ingrowth of secondary cementum which has reduced the lumen to ⅓ of its original size. Higher magnification, Fig. 10, will show this more clearly. Fig. 11—Case 14-E, from the same dog, but another vital case, shows secondary cementum extending the entire length of a lateral canal.

SUMMARY

These experiments are not only confirmatory as to the bacteriologic and roentgenographic evidence, but they demonstrate a point in which parodontists are undoubtedly most interested, that is, the potentialities of the parodontal structures to repair following adequate surgical treatment.

The regeneration of cementum and normal periodontal membrane should be of interest to both parodontists and root surgeons.

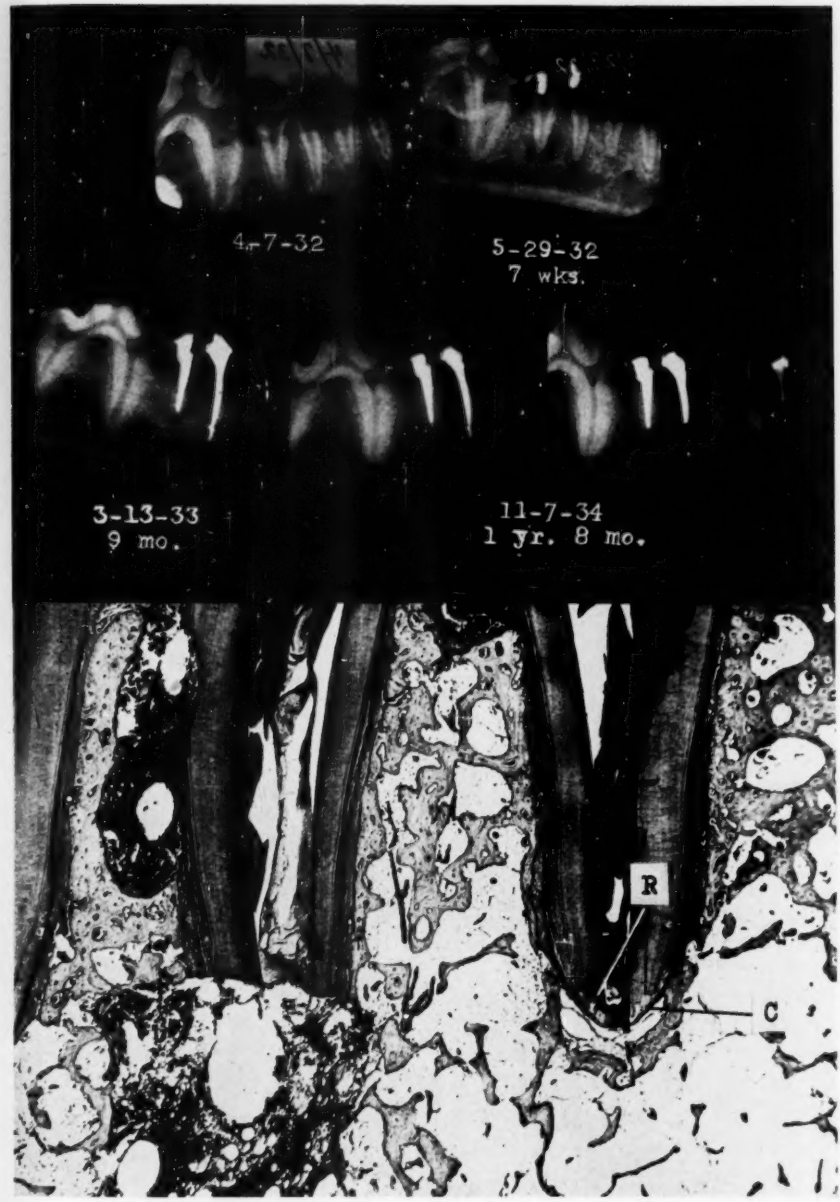

FIG. 8—Case No. 61-E. Infected case. Lower left molar 1 year 8 mo. after root canal filling. Mesial root was resected for bacteriological study (negative culture), distal root retained for histological study. Diagnosis (distal root): Mild chronic productive apical parodontitis showing newly formed cementum (C), beyond previous resorption (R). Above. Roentgenographs, No. 1, original; No. 2, rarefying osteitis about mesial

and distal roots seven weeks after inoculation; No. 3, taken immediately after root canal filling—pathosis quite perceptible about distal root and also around mesial; No. 4 and No. 5, taken before necropsy—before and after apecoectomy. The mesial root was resected for bacteriological study which was negative and the distal root retained for histological sections shown here. It is of interest to note the marked decrease in translucency as one compares the roentgenograph taken on 5-29-32 with 11-7-34, 1 year 8 mo. after root canal filling. This decrease in the rarefying osteitis as evidenced by the roentgenograph checks very favorably with the pathological diagnosis.

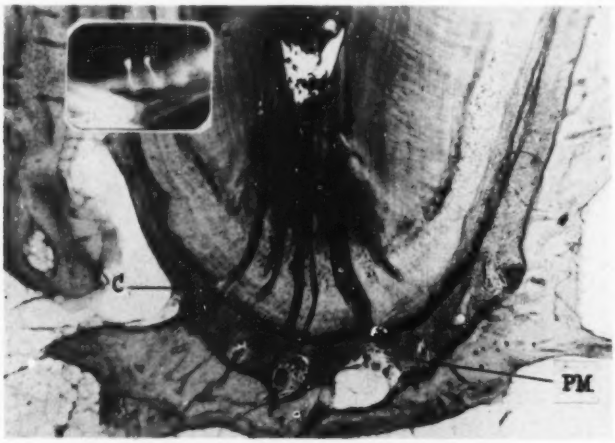

FIG. 9—Case No. 13-E. Vital case. Lower right molar (distal root) P. M.—normal periodontal membrane; (C) newly formed cementum obliterating ⅔ of lateral foramen. See high power Fig. 10. Insert. Roentgenograph showing root canal filling.

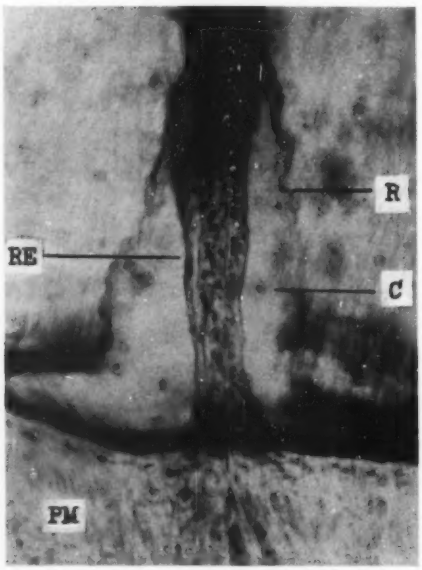

FIG. 10—Vital case. Higher magnification of Fig. 9. Showing obliteration of ⅔ of lateral foramen. (R)—resorption of original foramen; RE—remaining narrowed canal showing blood vessel within; C—cementoblasts; PM—normal periodontal membrane over apex of root (x550).

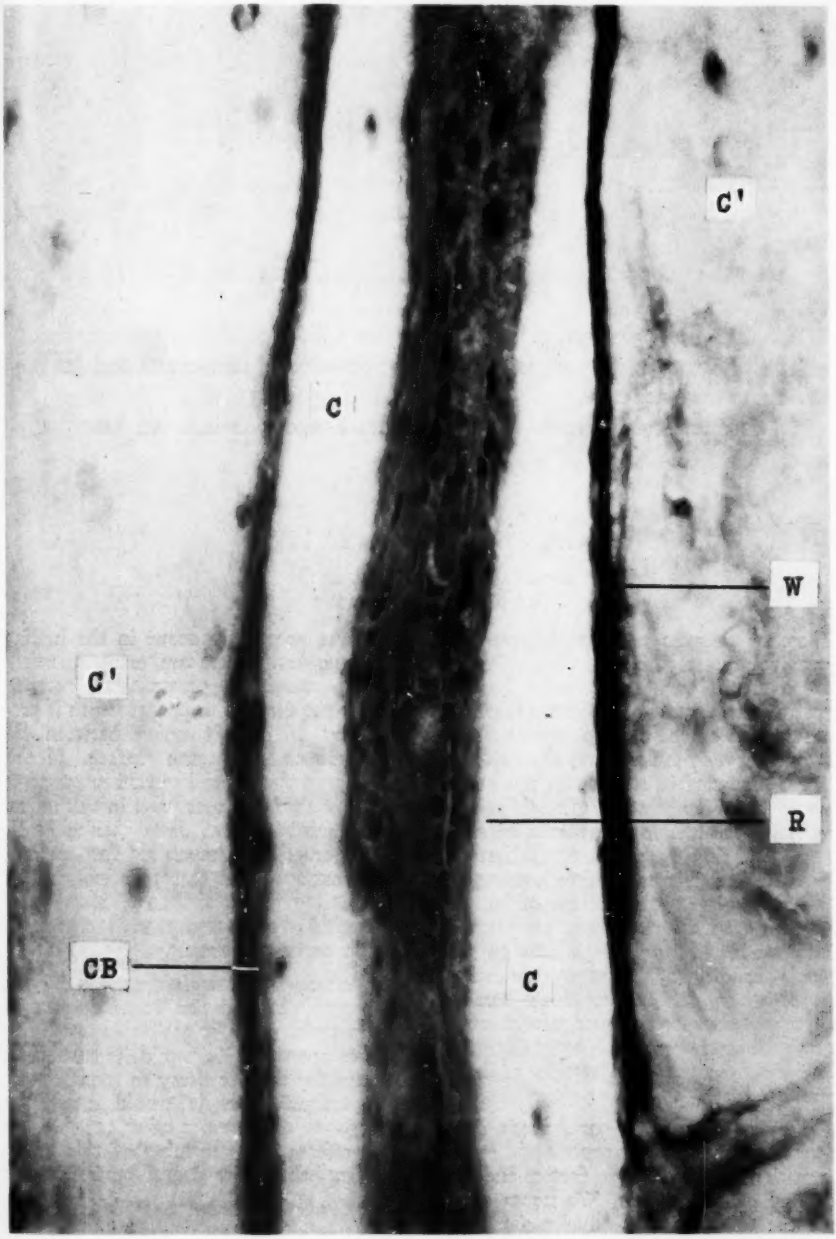

FIG. 11—Case No. 14-E. Vital case. Distal root of lower right molar showing ⅔ obliteration of entire length of lateral canal. C—cementum; W—original wall of canal; C—newly formed cementum showing cementoblasts (CB); R—remaining portion of canal (x550).

CONCLUSIONS

1. The use of the dog as an experimental animal for this experiment was justified.

2. Two types of cases were studied: the infected case and the non-infected, so-called vital case.

3. The infected cases showed a regeneration in kind by the manifestation of new alveolar bone, newly formed cementum, and a normal periodontal membrane.

4. The vital cases showed a normal periodontal membrane throughout the experiment.

5. Roentgenographic examination showed a diffuse rarefying osteitis about the apices of the infected cases, and normal periodontal membrane and lamina dura about the vital cases.

6. Bacteriological studies of both infected and non-infected cases were made.

A Case of Reattachment?

By Dickson G. Bell, D. D. S.
San Francisco, California

Those of us who treat paradentosis by subgingival curettage believe that it is possible to secure an organic union of the separated soft tissues to the cementum and that resection of the gingival tissues is seldom necessary. It is true that the evidence which is presented in support of this statement is based largely on clinical experience, never-the-less, it is evidence of such a character that it merits consideration. The scientific evidence to date consists of only a few microscopic sections of treated cases, and even these are open to question because of lack of sufficient data on the original condition. However, scientific evidence is very difficult to obtain because patients will not sacrifice successfully treated teeth for study purposes—especially since it includes removal of a section of bone around the tooth.

The research work in paradentosis during the past few years has contributed much information on the etiologic factors and the pathology of this disease. We know more about the tissue changes which take place in the paradentium, with special reference to the *formation of a pathologic pocket.* However, histologic information is most limited as yet about the *reparative* process that must necessarily occur in the healing of the pocket. There are many questions to be histologically proved, for instance, whether the decrease in pocket depth is produced by an organic union between the soft tissue and the tooth surface. If this really takes place, the question arises as to what part the blood clot plays in this union. Furthermore, in what cases such a repair may be practically impossible for one reason or another, and if we do not believe in such a biologic repair, what process takes place after gingivectomy with regard to the soft tissue attachment.

An attempt is now being made by the two dental schools in San Francisco to provide material for a scientific investigation of these questions. It would be a splendid objective for this Academy to sponsor such an investigation, as it would contribute greatly toward a solution of this problem—which affects every member of this group—in fact, the entire dental profession.

Our discussion must necessarily be limited to the clinical and roentgenologic information which is available at this time.

In the first place, we must consider our main purpose in the treatment of the

pocket. My observations yield the fact that pockets are usually closed completely as a result of subgingival curettage. They even remain in this status indefinitely—I have many such cases that have been under observation for over 15 years. No further treatment is necessary—only the usual periodic prophylactic measures. Subsequent roentgenologic check-ups have revealed not only an arrest of the marginal resorptive process of the alveolar bone, but also that a regeneration of bone has occurred in a number of cases—in a vertical direction. My first observation of the possibility of the deposition of new osseous structure, proved the clinical experience, that subgingival curettage produced most favorable results. Also, that by preserving the original organic matrix, the osseous part of the paradentium was strengthened.

This question of bone regeneration has been widely discussed. The practitioners who prefer the surgical treatment, apparently believe that this cannot occur, otherwise they would not destroy the matrix. Kronfeld, in discussing bone regeneration states "applying this principle to the bone beneath a suppurating pocket, it is certainly possible for the bone to regenerate after the cause of the bone destruction, namely the progressive inflammation of the underlying tissues, has been eliminated. This has been demonstrated clinically beyond any doubt. To what extent bone regeneration will take place in such a case depends, among other things, upon the amount of soft tissue attachment that has remained after the inflammation has subsided." T. Sydney Smith has published roentgenograms of a case in which 9 mm of bone had regenerated between the upper central and lateral incisors—in another, he states "7 mm of bone with an extremely thick cortical layer, having filled the space. The overlying gingival tissues were in an excellent state of health."

That bone regeneration can occur is by no means new. However, it must be emphasized that this regeneration can take place only in a vertical direction. A restoration of the cortical layer is the most that can be expected for those cases in which horizontal atrophy has taken place. A horizontal regeneration is therefore most doubtful.

May I present to you a case in which vertical regeneration has taken place in an elderly patient?

The picture on the left shows a pocket between the upper left lateral and cuspid before treatment by subgingival curettage in July, 1929—that on the right shows the same area in December, 1931 or 2½ years after treatment. The patient was 73 years old at the time.

Clinical examination showed an acute pericemental abscess on the labial surface of the cuspid—the tissue was elevated and there was drainage through a fistulous opening. The amount of tissue detachment on the labial surface was only 3 mm on both lateral and cuspid, 4 mm on the labial-interproximal—on the lingual, the interproximal depth of the pocket was about 4 mm on the distal surface of the lateral and 8 mm on the mesial surface of the cuspid; in other words, the opening of this deep pocket was on the mesio-lingual angle of the cuspid; it was rather narrow for a short distance and then suddenly opened into a very large area which extended over the labial surface of the cuspid. The interproximal soft tissue on the labial surface was slightly inflamed because of a small bit of calculus and some debris—whereas on the mesio-lingual of the cuspid, there was a decided inflammatory condition. There was an unusually hard mass of calculus on the mesio-labial angle of this cuspid—in fact, this patient had about the hardest deposits that I have ever attempted to remove.

Even though it is very difficult to observe remnants of the organic matrix, a slightly greyish line seems to indicate that

it is present in this case. Removal of this structure with interproximal curettes would doubtless have led to a complete destruction of this matrix without any possibility of future bone regeneration.

You will notice in the second picture that the instruments cut through the cementum into the dentine, and that this corresponds to the location of this nodule of calculus. As I mentioned, the deposits were so hard and so firmly attached to the cementum that they could not be easily broken away with the instruments, but had to be gradually planed off. The fact that the pocket was on the mesio-lingual and the calculus was on the mesio-labial also made it very difficult. One lesson learned from this case was, that a small incision should have been made at the point of the fistula, thus giving an opportunity of treating the pocket from the labial surface as well as from the lingual, and undoubtedly this cut into the dentine would not have occurred.

However, you will observe that the regenerated bone has arched over this area—it is not as close to the tooth at this point as it is below it. It is possible that the accidental cutting into the dentine is responsible for the lack of complete filling in of the bone. It is also possible that part of the organic matrix was severely damaged near the tooth through excessive instrumentation, or perhaps by the inflammatory process, so that a complete restoration could not take place. The last examination of this treated area showed marked improvement in this gingival condition. There was no discharge and the abscess had healed completely. Whether the tissue has become attached to the surface of the tooth, or only adapted to it, cannot be definitely proved without histologic sections. However, clinically, the condition appeared to be most favorable from the standpoint of gingival recovery and bone deposition.

You will probably understand now why I do not favor any surgical procedure which calls for the removal of the organic matrix, parts of the alveolar structure, or even any of the gingival tissue.

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—Extract from Subscription Leaflet.

Radiology in Prevention, Diagnosis and Treatment of Periodontoclasia*

By GEORGE R. WARNER, M. D., D. D. S.
Denver, Colorado

THERE is an old saying, "An ounce of prevention is worth a pound of cure," with which we are all familiar, but which is so apropos in the consideration of periodontoclasia that I want to use it as an introduction to this paper.

At first thought the use of radiology in the prevention of periodontoclasia seems rather far fetched. However, on maturer consideration, we realize that we can perhaps more clearly see the beginning of pathological changes in the periodontium with the aid of radiology than with the eyes alone.

Prevention. But, one may say, if pathological changes in the periodontium have already taken place just where does "radiology in prevention" come in? A pertinent question. But if the very early evidences of periodontal involvement could always be recognized we would be much further along in controlling periodontoclasia than we are at present. However, let us go back to an earlier period, when no recognizable changes have taken place in the supporting structures of the teeth. Is radiology of help then? It should be. Morgan (1) says "A vast amount of knowledge of the teeth and jaws of a child can be gathered from roentgenographic studies made during the growth and developmental periods." At this time we can see, with the aid of the roentgen ray, the size and shape of the tooth roots. Perhaps we can gain an even better idea of the shapes of the tooth crowns, contour, and interproximal spaces, than we can by a clinical examination alone.

We can early adopt appropriate preventive measures if the " 'teen" age radiographs show short or peg shaped roots, missing teeth, malocclusion or other conditions predisposing to periodontoclasia.

Orthodontia plays an important part in the prevention of periodontoclasia and radiology is indispensable in the practice of this specialty of dentistry, as brought out years ago by Albert H. Ketcham. (2) The extra oral radiograph, disclosing as it does the position of the third molars, is valuable in preventing periodontoclasia by pointing early to the necessity of removal of third molars that are tending to force the remaining teeth into bad occlusal relationships. Moreover, if third molars are so positioned that removal is advisable or inevitable, it is essential to have them removed early enough that the alveolar bone will build in on the distal aspect of the second molars and thus prevent a pocket that not infrequently results in the loss of the second molar.

The use of radiology indirectly aids in preventing periodontoclasia by disclosing interproximal caries early enough that measures may be instituted that will prevent loss of mesio-distal diameters of teeth with consequent invitation

*Read before the American Academy of Periodontology at San Francisco, California. July 11, 1936.

to periodontal disorders through packing of food against the septal soft tissues and interfering with normal circulation.

The foregoing is especially directed to the prevention of periodontoclasia in early life but the same measures are applicable in later life. Radiology is helpful in discovering faulty man-made contact points, over and under contoured fillings which pinch soft tissues or allow food impingement. An over generous amount of filling material left under the gingival margin is sometimes difficult to discover without the aid of the roentgen ray. Mother Hubbard gold crowns can be discovered in a clinical examination, but how much more one is impressed with their potentialities for harm to the periodontium when seen in a radiograph. Damage to the periodontal membrane from undue stresses can be recognized so clearly and early in a good film that the preventive value of radiology would be established for such cases.

Just where subgingival calcareous deposits come into the picture in periodontoclasia we are perhaps not all agreed. Even the exact starting of periodontoclasia is a matter open to discussion. But it seems to me we may have a simple gingivitis from bad dental restorations, as partially enumerated above, and also from calcareous deposits. If this is true the depiction of such deposits or such faulty restorations in a radiograph would put radiology in the role of a preventive agent in periodontoclasia; for such deposits or such faulty restorations causing a gingivitis which later develops into periodontoclasia would have to be considered an active factor in that disease.

Diagnosis. In diagnosing periodontoclasia in the days before we made a great deal of use of radiology we placed much stress on the condition of the gingiva. We spoke of its position, form and color, as being of diagnostic significance. We still look to the gingiva for aid in diagnosing periodontoclasia when we make our clinical examination, but I am sure no one of us would care to depend entirely on the clinical examination in diagnosing this disease since we have learned to depend on radiology as an aid in diagnosis. Dr. Clarence O. Simpson (3) says, "Excepting superficial lesions, radiodontic examinations will supply about 80% of the evidence for dental diagnosis."

If we are to use radiology in diagnosis I am sure we are agreed that we should make full use of it. By that I mean we should thoroughly cover every area of the mouth with properly positioned and skillfully processed intra oral films and have them mounted in proper relation to one another in a light excluding mount. Such a set of roentgen-ray negatives makes for clarity of thought in interpreting the films and evaluating the conditions noted. This is shown beautifully in "Roentgenographic Studies of Periodontal Disease," by Henry Cline Fixott. (4)

Many of us are so situated that we must depend upon ourselves to produce such a set of radiographs. It is therefore encumbent upon us to develop the

necessary radiologic technic as a part of our preparation for the treatment of the disease under consideration.

With the mount properly illuminated we should study the films with the idea of determining causes and effects.

Can we learn anything from the character of bone trabeculation, either as to the arrangement of the trabeculae or the mineralization? If we can I must confess ignorance of the significance of particular types of trabeculation or amount of bone earth as shown by resistance to penetration of the ray. Certainly radiolucence or radiopacity of bone, *per se*, is not indicative of predisposition to, or immunity from periodontoclasia.

Boyens, (5) in an article on "Etiologic Factors in Various Types of Paradentosis," gives illustrations of bone changes with which we are all familiar, such as the horizontal and vertical types of periodontoclasia; in both of which conditions he says the bone is resorbed from without. He then gives illustrations of dystrophic types of alveolar bone in which the bone resorption is from within. It is possible, or perhaps I should say probable, that more intimate knowledge of bone pathology and the roentgenographic depiction of pathologic conditions in bone will enable the periodontist to interpret his intra oral films better than at present. But at the present time I do not believe we can safely depend upon radiology to make the fine distinction in types of bone structure that would indicate to us a susceptibility to, or an immunity from periodontoclasia. In an article entitled, "Observations on Periodontoclasia, With Special Reference to Autogenous Vaccines and Surgical Intervention," Lose and Fourè (6) say "Prominent alveolar recession was present in all cases and affected particularly the *lamina dura* of the interdental septa, as far as could be determined by radiograms, and the cancellous bone was of spongy consistency." On the other hand Kaushausky (7) calls attention to the fact of "hypercalcemia in pyorrhea cases" a condition which I am sure we have all noticed and in keeping with the old saying, "Pyorrhea is a disease of good health." Kronfeld (8) holds that, "the only changes found in the alveolar bone in the vicinity of pyorrhea pockets are those of osteoclastic resorption."

W. A. Colburn, (9) in an article entitled, "Classification and Roentgenologic Interpretation of Dental Surgery" says under "pyorrhea or parodontitis," "This condition is easily recognized by the looseness of the teeth, and in a majority of cases the condition is attended by a flow of pus from the margins of the affected alveolus, and by deposits of calculi on the sides of the denuded roots." But, while in other parts of the article he talks about alveolar atrophy and osteomalacia, and illustrates with beautiful roentgenograms, he says nothing about the appearance of the alveolar bone in parodontitis.

Pollia (10) says, "The use of the radiograph gives the most definite and the earliest information regarding the condition of the supporting bone tissue, and

is, without doubt, the most powerful factor in the prevention of extreme changes, provided it is used with the frequency which certain cases may demand." However, he says nothing about the alveolar bone, in a case of periodontoclasia, having any particular characteristics. To be sure he calls attention to the changes which take place adjacent to the teeth, changes which we all recognize.

So let us examine our radiographs for those causes of periodontoclasia which we can all recognize.

Under etiology, in an article on "Periodontoclasia, Etiology and Treatment," Merritt (11) says, "In the second group are to be found all those conditions that interfere with the normal function of the teeth and jaws. These are lack of vigorous use in mastication, malocclusion, loss of teeth, food impaction, trauma, etc." Lack of vigorous use in mastication would be evidenced in the radiograph by unworn cusps, and thin alveolar crests. Malocclusion is not so evident in intra oral radiographs unless we have bite wing exposures. Loss of teeth is, of course, evident in the intra oral film and the amount of malposition due to loss of teeth is probably more evident radiologically than clinically. While food impactions cannot be seen radiographically the cause for such impactions is made most evident by this means. Trauma can usually be more graphically depicted by the radiograph than any other means. "Et cetera" is the final thing listed by Dr. Merritt, and, as he will agree, this covers quite a long list of causes, such as: overhanging fillings, fillings with faulty contour, artificial crowns with bad contour, with too great an occlusal area, or with an area too large for the tooth cervix or with band extending too far under the gingiva. Dental caries under fillings and on proximal surfaces of the teeth are factors in causal relation to periodontoclasia. (12) Peculiar shape of tooth crowns; and certainly short peg shaped roots, are inviting to the destructive action of periodontoclasia. Then the most common of local causes, calcareous deposits, are shown so clearly in a roentgenogram that even the untrained eye can see them.

The *effects* of periodontoclasia are probably more clearly shown by radiology than clinically. The destruction of alveolar bone, the widening of the peridental space, destruction of the lamina dura, extrusion and drifting of teeth, all stand out with cameo like clarity in the radiograph.

Treatment. The discernment of etiologic factors in periodontoclasia through the medium of radiology is most helpful in the treatment of the disease by both preventive and remedial measures. The amount and position of bone destruction, the appearance of the lamina dura, the width of the peridental space at various points on the roots of the teeth, the shape, length and number of roots of the various teeth; these and possibly other diagnostic entities are so clearly shown in the radiograph that treatment seems hopeless without its help. This

is clearly brought out by McCall (13) in "The Radiogram as an Aid in the Diagnosis and Prognosis of Periodontal Lesions."

It is said that a correct diagnosis constitutes the major part of the treatment of disease. But while radiology is most helpful in the diagnosis of periodontoclasia it is perhaps not much less helpful in its treatment. By the clear depiction of the depth of pockets the operator has a guide for subgingival curettage or more radical surgical procedures.

As we all know, it is essential in the treatment of this condition to free the subgingival surfaces of the roots from all calcareous deposits. Strean (14) says, "Irritation is generally accepted as one of the primary etiological factors of periodontoclasia." It so happens that it is quite difficult to remove these deposits completely and it certainly is not easy to prove by sense of touch that such deposits have been completely removed. The most resistant of these subgingival deposits are on the proximal surfaces of the molar and bicuspid teeth. By the simple expedient of two bite wing exposures the matter of residual subgingival deposits on molars and bicuspids of both jaws can be pretty definitely settled.

As occlusal trauma can usually be demonstrated radiologically so also can success in its treatment be checked by the same means.

Failure in treatment is all too clearly shown by the radiograph, and, unfortunately if we wait too long in checking our results our poor results cannot be rectified. Success in treatment is not always shown radiologically by improved conditions but rather by conditions remaining status quo. As a matter of fact the cases that show no favorable changes, as depicted by the radiograph, do often show favorable changes clinically. And, if we were keener in interpretation of our films, we might see favorable changes therein that we do not now recognize.

As the success of treatment of periodontoclasia depends on the patient's coöperation and as the beginning and continuance of that coöperation is at least partly dependent upon a clear understanding of conditions by the patient, radiology is most helpful in giving the patient a clear picture of the disease in the beginning and as the treatment progresses. If, as it not infrequently happens, the patient does not respond to promised periodic calls, the radiograph is helpful in making clear to him the evil results of his indifference or procrastination. Extrusion of teeth which are out of occlusion is sometimes more noticeable in the radiograph than in the mouth. Drifting of teeth is sometimes brought to our attention by the radiograph when it had passed unnoticed in our periodic treatments and conversely patients sometimes get the notion that teeth are changing position where follow-up radiographs show there has been no change. By this most convincing ocular evidence our position as dentists is strengthened and the value of radiology enhanced in our patients' minds.

In spite of the great and growing value of radiology in the periodontist's

armamentarium we must recognize its limitations. W. J. Charters (15) points out that destruction of periradicular bone may be deeper than indicated by the radiograph.

Rudolph Kronfeld (16) goes so far as to say " . . . the usual procedure of diagnosing 'pockets' from a radiograph is incorrect." Dr. Kronfeld brings out the point that "pockets" may not be as deep as indicated in a radiograph and illustrates his text with a radiograph and photomicrograph of the same area.

The careful periodontist is not likely to fall into either one of these pits, because in diagnosis and treatment with a radiograph to aid him, he can depend on his fine sense of touch to guide his instruments to the bottom of pockets but not beyond.

So, I hope you will agree with me that radiology is of inestimable value in the prevention, diagnosis and treatment of periodontoclasia.

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Nutrition and Malnutrition from the Laboratory and Clinical Aspects*

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IT has been proved in many laboratories that dietary deficiencies of calcium and phosphorus, as well as vitamins, account for abnormalities in structure of bone and tooth. Furthermore, when experimental animals such as rats are maintained on a diet consisting of cereals, peas, and beans, serious skeletal defects ensue, and even the addition of muscle meat is not sufficient to overcome them. In addition, these monkeys, rats, guinea pigs, and dogs present dental lesions such as hypoplasia, caries and gingival resorption, which are somewhat comparable to human dental pathology. However, it is difficult to correlate the various dietetic results obtained with different types of animals for the reason that some of them, for example, rats, are able to synthesize one of the vitamins—cevitamic acid (formerly designated vitamin C or ascorbic acid). Likewise, the guinea pig is not so susceptible to vitamin A deficiencies as the rat. Until correlations between these experimental animals have been completed, it will be difficult to draw conclusions from these results of dietary deficiencies which can be applied to the etiology and symptomatology as observed in the human being.

In spite of all this, some investigators believe that results secured from feeding guinea pigs a diet deficient in cevitamic acid proves a direct connection between scurvy and pyorrhea. When experimental animals are maintained on a strictly "scorbutic" diet, the teeth loosen in about three weeks, the gums become spongy and bleed easily; later the dental pulps undergo degenerative changes. Several investigators have produced extensive alveolar resorption by this method. Gingival pockets and lesions similar to pyorrhea have been observed in which the nutritional deficiency was the apparent cause. But this is not a true pyorrhea, for the inflammed oral mucosa quickly responds to a corrected diet by returning to normal. That food has a definite relationship to alveolar resorption in the human is an hypothesis which has been established only in part. To produce malnutrition in some species of animals is a daily laboratory experience; in others it is so difficult that few have had success. However, to translate into terms of clinic application the results of laboratory experiments, requires the combined efforts of clinician, pharmacologist, and biochemist.

It is pertinent here to draw attention to the fact that in the development of the science of nutrition, progress has been made by discussion of new ideas; but when the arguments became bitter and personal, the advantages of such polemics were nil. There have been many instances in which the enthusiastic worker has been maneuvered into a false position and has been charged with statements which, if made, were misinterpreted by others. From the standpoint of argumentation or debate, such is the accepted procedure, but to contradict facts by

*Read before the American Academy of Periodontology, San Francisco, California, July 10, 1936.

quarrelsome methods, is egotism in the superlative and disqualifies at once the disputant. For example, the studies by Liebig, Voit, and others upon the importance of protein in the dietary was argued with true Teutonic bitterness and singleness of purpose. However, as in any debate of importance, both sides were partly correct; neither was entirely wrong. Similarly when calories were first determined for various foodstuffs, the utilization of carbohydrate, fat, and protein again was the subject of bitter controversy. One needs, at times, the ability to see around the corner, so that the various interpretations of the data may be given their true value. A fact needs background and foreground just as a figure in a picture. Only by understanding the development of certain ideas can the facts, crystallized and confirmed from the laboratory, be understood and accepted. To be more specific, consider statements made less than fifty years ago about the composition of bone.

One of the earlier descriptions of bone defined it as a tissue consisting of water and about equal amounts of inorganic and organic material. This now is obsolete because it is inadequate. It has been established that bone consists of an inorganic portion comprised mainly of calcium phosphate and carbonate in an approximate proportion of three to one. In addition there occurs, also, small amounts of magnesium, chlorine and fluorine compounds. The frequent attempts to discover the manner in which these various salts are bound to one another indicate the degree of uncertainty in the minds of many workers in this field. The suggestion was made many years ago by Hoppe-Seyler that the chemical structure was similar to a mineral, apatite. Later this idea was partly supplanted by another hypothesis to the effect that the mineral dahllite is more closely akin to bone. However, the recent work of Janet H. Clark,[1] at Johns Hopkins supports the older view. By applying X-ray diffraction methods, new facts relative to the structure of bone in health and disease have been presented. To put a complicated matter briefly, the thesis essentially is this:

(1) Inorganic bone crystals are so placed as to present a definite fibre structure in longitudinal section.

(2) Crystals not so placed are organic in nature and are derived from collogen or ossein.

Confirmation of these views comes with a published report on rickets from the University of Illinois by Clark and Mrgudich.[2] The longitudinal arrangement of the inorganic particles of bone is destroyed in rickets partly by reason of the breaking down of the organic cement. This results in a weakening of the architecture of the inorganic material and bone resistance to strain is lost. The supporting bone of the alveolar structures, likewise, probably undergoes similar changes. Furthermore, Clark and Mrgudich[2] suggest that any theories which consider an equilibrium between serum and bone calcium should include similar equilibria between blood serum and bone organic matrix.

The factors, both dietary and endocrine, responsible for the formation and maintenance of bone and tooth structure are subjects of daily discussion, and, if their relation were as simple as sometimes appears, malnutrition would cease to be a problem of major importance. The number of statements made, and almost in the next moment refuted, have served to confuse rather than clarify some parts of this research. In the earlier days of vitamin controversy, not the least of these difficulties was in the nomenclature. But as the chemistry of vitamins is being solved, the nomenclature, likewise, becomes somewhat more simplified.

Drug houses and similar pharmaceutical manufacturing concerns, anxious always to follow closely the research in medical science, sometimes in their enthusiasm, get a step or two ahead of controlled research and thus put the patient in jeopardy. Consider merely the recently determined facts in connection with the indiscriminate use of ergosterol as an agent to promote health by reason of its vitamin content.

There is even greater activity as well as confusion in the field dealing with laboratory and clinical studies of thyroid, parathyroid, pituitary, and other ductless glands. Earlier experimental work upon the pituitary, for example, consistently indicated that this gland, at least in dogs, and probably in man, is not only essential to life, but controls, also, the complex mechanism of calcium metabolism. This theory has been elaborated by the experiments of Dandy and and Reichert[3] on hypophysectomized dogs. They have so developed the technique that they have succeeded in removing the pituitary and in avoiding the fatal consequences which, heretofore, have been characterized as sequelae to these operations. The authors suggest that the very marked symptoms, formerly described as developing from the removal of the gland, arise from factors other than loss of the pituitary. These are summed up under the term, "cerebral trauma" incident to the operation. The studies of Evans[4], Simpson, Smith, Collio, and many others indicate that the pituitary controls, to a large degree, not only rates of skeletal growth, (both dwarfism and giantism have been produced in experimental animals) but exerts also an important regulatory effect upon the organs of reproduction and lactation. In addition, it is closely associated with thyroid, parathyroid, and adrenal activity, and with the metabolism of fat, carbohydrate, and probably protein.

In connection with rickets which arises from deficient diet, interesting observations have been offered on the increased size of the parathyroid glands. These organs, when examined by post mortem methods, show a distinct enlargement. Analogous changes in the human being have been found in patients suffering from an abnormal growth of the bony structure. The explanation suggested is that the rachitic disturbance in calcium metabolism is being partly compensated for by an hyperactive parathyroid.

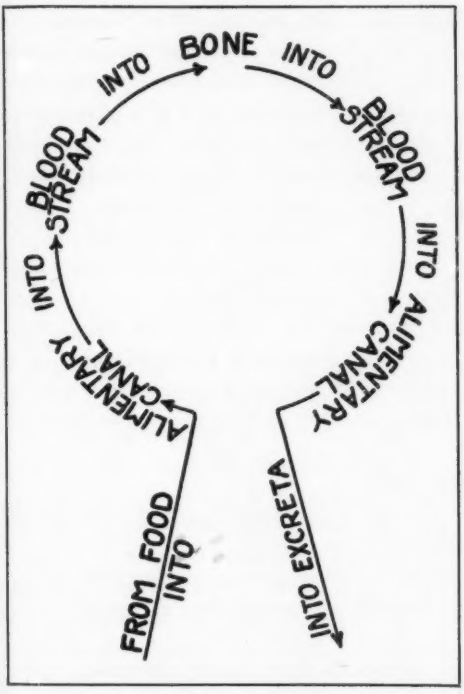
In addition to the calcium and phosphorus compounds inevitably associated with nutrition of bone and teeth, there are four other chemical elements which play a very important part in the whole study of bone and tooth formation. They are the four members of the halogen group of elements—the *four horsemen,* so to speak, of calcium and phosphorous metabolism and therefore, variously concerned in the development and maintainance of normal bone and tooth structure. The chloride ion is a normal constituent of all tissues. It is present in the stomach in the form of hydrochloric acid and constitutes an important part of gastric digestion, and hence of utilization of foods. In the blood stream it is present as the soluble salt, calcium chloride, and supplies the lime which later is precipitated as the insoluble calcium phosphate. The iodide ion, found mainly in the thyroid gland is another factor in the utilization of calcium and phosphorus by the body.

The bromides are adventitious substances. When, and if, they are found in the tissue, it is usually in individuals who have been on a therapeutic regime which included bromides—usually the sodium or potassium salt. Their function, therefore, in calcium metabolism is not clear.

It is possible that fluorine compounds are present more by accident than by metabolic necessity. The fluoride ion exerts a deleterious effect when present even in small amounts above the physiological optimum. The development of mottled enamel in experimental animals by feeding fluoride has been reported by Smith and confirmed by De Eds. This has a most important bearing upon both osseous and dental growth. But there is a gap in the story, for no one yet has shown that alveolar structures are adversely affected by fluorides. Such a supposition is not improbable. Machle[5] points out that there exists presumably a normal metabolism of fluorine, and, until the degree of normal elimination can be determined, it will be difficult to estimate the physiological optimum and its relation to daily fluorine intake or out-put.

In addition to the various dietary and glandular studies on nutrition and utilization of inorganic salts, there must be considered also the local agents participating in bone growth and in changes constantly occurring in osseous tissues. The first of these depends upon the relation of blood supply to the ossifying tissues. The second deals with the mechanism of precipation reactions by means of which the soluble lime salts are converted into insoluble ones. During the process of bone formation the rate of flow of blood and lymph is slowed down. Thus, a point of minimum flow is reached and a condition established favorable to deposition of lime salts. A deficiency in blood calcium at this point interferes with the process. Here a further confusion arises. It has formerly been assumed that the chemical determination of blood calcium provided an index of the amount of lime entering the bone. This directional flow of inorganic salts is indicated in the upper half of the chart.

To determine merely the amount of lime salts in the blood stream, as is being done, is hardly a fair index of the direction of flow. The arrows may be pointed exactly in the reverse direction. Instead of lime being deposited in bone, it may be leaving it, as is diagramed in the lower segment of the drawing.



It is well known that the mineral parts of bone are not merely inert masses, unlikely to be changed or disturbed after they have been deposited. Indeed, the opposite is the case. These mineral substances are constantly involved in the interchange of inorganic salts which occurs between the circulatory fluids, the bone and other tissues and organs. In fact, bone, rather than skeletal muscle serves as the store house for lime. It is only called for in case of calcium deprivation such as arises from deficient diets or glandular dysfunction.

The second factor in this local tissue change deals with the deposition of calcium phosphate. It has been demonstrated by Kay and his co-workers, as well as by Bodansky and many others, that during the formation of bone and tooth structure, there is present in the area a substance which accelerates the deposition of the inorganic salts. The complicated reactions by which this result is accomplished have been described elsewhere, but it is established that the precipitation of tricalcium phosphate is governed by the enzyme "phosphatase." Recently, Jacobson[6] has demonstrated a new specific phosphatase which utilizes blood corpuscles as a substrate. In addition, it presents definite chemical properties which differentiate it from the phosphatase of tissue.

The phosphatase theory serves not only as a possible explanation for normal tooth development, but it may apply also to the mechanism of mal-formation of dental structures. It has been demonstrated by several laboratories that mottled enamel is associated with the presence of fluorides in drinking water as well as in food. Furthermore, it has been stated that fluorides may, in certain concentrations, increase the activity of phosphatase, the enzyme. If this be the case, there are two suppositions possible: first, the activity of the phosphatase is

in the reverse direction, thereby producing resorption of bone or tooth substance; second, the accelerated activity of the enzyme brings about the rapid formation of insufficiently calcified or hypoplastic areas. De Eds has noted that fluorides, instead of increasing phosphatase activity, as is claimed by some investigators, exert an opposite effect and reduce the potency. In either event, there seems to be no reasonable doubt but that compounds of fluorine influence, not only the formation, but also the resorption of normal calcified tissue. It may be postulated that the resorption of deciduous roots from growth stimulus, or even of permanent roots from injury, by producing a localized hyperemia, brings the enzyme into the era, and the acidotic reactions characteristic of injured tissue may tend to activate the phosphatase in the opposite direction. Therefore, instead of building tissue, it tears it down by resorption. Recent studies on rickets provide further light upon this subject. Smith and Maizels have demonstrated that in rickets, phosphatase is present in amounts over three times that found in normal growing bone, and suggest that it is derived from the greatly proliferated cartilage cells and osteoblasts. Robinson has shown that osteoblasts are one source of the enzyme, so its presence in increased quantity is a necessary factor in ossification. This theory of phosphatase action may also be applied in part to the formation of mottled enamel, as well as to other types of dental hypoplasias.

SUMMARY

Nutrition and malnutrition from the aspect of skeletal growth and health maintainance may thus be summarized:

First, a dietary intake which is adequate in all respects, and especially in the amounts and proportions of available calcium and phosphorus compounds and vitamins.

Second, the efficient utilization by the tissues of these various substances. This is the more complex of the two and includes the following factors:

(a) the pH values of blood plasma and intestinal contents.

(b) the amount in blood plasma of:

1. ionized and non-ionized calcium,
2. dialyzable and non-dialyzable substances,
3. phosphate compounds.

(c) abnormal or normal amount of parathormone.

(d) the degree of activity of the thyroid gland and its influence upon basal metabolism.

At present, there is insufficient evidence which would justify definite statements upon the relative importance of these different items. Although they control the local activity in calcifying areas to a large extent, there are, in addition, certain cellular elements to be studied, such as the osteoblast and catalytic agent of calcification, "phosphatase."

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The William J. Gies Testimonial

By ARTHUR H. MERRITT, D.D.S., F.A.C.D., F.A.A.P.
New York City

FOR twenty-eight years a man who is not a dentist has given of himself, his time and his talents, to the advancement of dentistry. To this self-imposed task he brought unusual qualifications—a scientifically trained mind, great executive ability and a prodigious capacity for hard work. And when to these are added a personality that invests all he does with a spiritual quality that is as unique as it is rare, plus the vision of a great ideal, one begins to understand something of the spirit that has animated this man in his unselfish service to dentistry.

For more than ten years, he carried on extensive researches in dentistry along many lines; in 1919 he founded the Journal of Dental Research, the value of which in the progress of dentistry cannot be overestimated; as a further incentive to dental research he organized, in 1921, the International Association of Dental Research which is at present operating through twenty-six sections, nine of which are in foreign countries; from 1921 to 1926 he was engaged in making a survey of dental education in the United States and Canada, a report of which was published in a volume of over seven hundred pages. And this is only a partial list of his many activities, all of which he has carried on, often at great personal and financial sacrifice, in order that humanity, through the profession of dentistry, might be better served. It is an amazing record, and one that is without a parallel in the history of dentistry. If there be those who cannot understand how a man can give so much of himself to a profession of which he is not a member, it is because they have not felt the driving force of a great ideal, nor known the satisfaction that comes from unselfish service.

And now, after the lapse of all those years, crowded with achievements in many fields, the dental profession is to do honor to this man to whom it owes so great a debt. This it proposes to do by giving to him a testimonial dinner at Atlantic City on Sunday evening, July 11, to which the entire profession is invited. This is being sponsored by the American College of Dentists with the cooperation of all national dental organizations. It promises to be one of the outstanding events in the history of dentistry—a spontaneous and enthusiastic coming together of the members of the dental profession of the United States and Canada to do honor to one of its great benefactors.

Endowment Fund for Journal of Dental Research

As a further tribute to Dr. Gies and as tangible evidence of the high esteem

in which he is held by the profession, it is proposed to raise an endowment fund of $50,000 for the Journal of Dental Research. For seventeen years of its existence, its founder has not only edited and prepared it for publication without compensation, but in addition has had the responsibility of making up each year an average deficit of $2,000 no small part of which he has paid out of the modest income of a college professor.

The raising of this Fund is to be undertaken along two lines: first, by increasing the number of subscribers to the Journal, and second, through direct contributions to the Fund from individuals and dental organizations, as set forth in the following plan:

(a) Classes of Subscribers:

(1) Annual subscribers: those who subscribe to the Journal annually, paying for it the full subscription price of $5 a year.

(2) Sustaining subscribers: those who agree to subscribe and pay $10 annually.

(3) Endowment subscribers: those who subscribe and pay $25 annually, $20 of which shall go to the Endowment Fund.

(4) Life subscribers: those who make a payment at one time of $100, the entire amount to go to the Endowment Fund. Endowment subscribers, at the expiration of four years as such, may, on request, be transferred to this class. Sustaining subscribers may be thus transferred at the expiration of ten years.

(b) Contributors to Endowment Fund. In a further effort to increase the Endowment Fund, the Committee proposes, in addition to obtaining individual donations, to invite the different dental organizations of the country to become contributors to the Fund. These contributors are to be of four classes: (A) Goodwill contributors: individuals and organizations contributing any amount up to $250. (B) Donors: those making a payment of $250. (C) Patrons: those contributing $500. (D) Benefactors: those who subscribe $1,000, or more. (Payments to be made in installments when desired.) Contributors of $100 or more, may, on request, be given life subscription to the Journal of Dental Research. In putting this plan into effect, an effort is being made to get in touch with every member of the dental profession through his State dental society in the belief that he will welcome an opportunity to have a part in preserving for dentistry one of its most valuable assets.

Never, perhaps, in the history of the profession, has it been given a greater opportunity to prove its right to be regarded as a scientific profession—to show by its support of this testimonial to Dr. William J. Gies that it is worthy of the faith which he has shown in it throughout all the twenty-eight years in which he has labored for its advancement. And there is no better way in which this can be done than by a united and wholehearted support of the Journal of Dental Research.

The Prevention of Periodontoclasia*

By HAIDEE WEEKS, D.D.S.
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IT SEEMS almost needless, perhaps rather presumptuous on my part, to express my appreciation of the honor that you conferred when you made me President of the American Academy of Periodontology. One must accept such an honor with humility, and with a keen realization of the responsibility it carries and realize that he cannot discharge perfectly the duties assumed and may be called upon frequently to make decisions that will not be approved.

My best efforts have been given to the task at hand, and the duties of this office have been discharged to the best of my ability and may I add that every member who was asked to serve on a committee accepted, and served efficiently.

In the last several years there has come a decided shift of values; more than ever we are turning to the things of the spirit, to intangible qualities. I am glad to say that we of the American Academy of Periodontology are rich in shared enthusiasms, in enduring friendships, and beautiful memories. These are the expressions of those imponderables—Life, Love and Death.

Those of us who saw Maeterlinck's "Blue Bird" will never forget the scene where the two children are near the little cottage in the wood of the "Land of Memory," where Granny and Grandfather sit sleeping in the warm sun. Suddenly they stir. Someone on earth is thinking of them and they happily awake from the peaceful sleep of death. To-day in the "Land of Memory" Sidney J. Rauh and R. S. Towne, those friends and companions who left us during this last year, shall be reminded now that we are thinking of them, and they shall be united to us by that bond of memory.

From the Preamble of the Constitution and By Laws of the Academy of Periodontology, dated February 21, 1914 the following is quoted: "We deem it for the best interests of the public and the profession that a society should be formed, to the end that those especially interested may meet and work together without prejudice for the scientific investigation of dental caries and periodontoclasia; that the practice of Oral Prophylaxis and Periodontia as an exclusive study be encouraged." From the Constitution, Art. II: "The object of the Academy shall be to promote and advance the science and art of dentistry as it pertains to the prevention and treatment of periodontal disease; . . . to encourage the study of the relationship of periodontology to general medicine and hygiene; to provide for the dissemination of knowledge concerning it and related fields of science."

As my life's work has been the prevention of dental and periodontal disease, and the above quotations assure me that I am "within the law," I have chosen for my topic the *Prevention* of Periodontoclasia.

* President's Address, read before the American Academy of Periodontology, July 9, 1936, San Francisco, California.

I am sure that some of us thought, when we left dental school that if we could get the child in time to save the first permanent molars, we were doing all that God expected us to do in this life. At least that is what I thought and I adopted for my slogan, "Give each child the birthright it so rightly deserves, four opposing molars."

How startled many of us were when we went forth to save the first permanent molars as one of our everyday duties, to discover that many times before the child was six years old, there were many abscesses caused by deciduous teeth, and the first permanent molars frequently presented themselves hypoplastic and badly formed on eruption.

How could we save a tooth that came into the mouth imperfect? We, of course, remembered from our embryology, that at the seventeenth week of foetal life the buds of the first permanent molars had their beginning, and that anything which caused a nutritional or metabolic disturbance in the mother caused a disturbance in that part of the foetus being formed, and so on through the life of the foetus, and subsequently through the life of the child.

Prevention, as we know, was practiced hundreds of years ago by the Chinese who paid their doctors to keep them well. It has taken us many decades to appreciate that fact and even now do we all practice it? Can we prevent by seeing our patients only when the ravages of disease have impaired their constitutions and the hard and soft tissues of the teeth beyond repair.

In my opinion, we should begin our work of prevention among the pupils in high school and should teach the prospective parents of our future generations the nutritive value of foods necessary to the building of a healthy body; the value of mother's milk; the necessity of fresh air, sunshine and exercise. A toothbrush should be used at the eruption of the first tooth and at eighteen months the baby should become acquainted with the dentist.

In the light of our present knowledge of nutrition we know that a child who is fed milk, cod-liver oil, and orange juice during infancy is usually protected against rickets and other deficiency diseases. A child, so fed, will have teeth that are more perfect and more resistant to decay, and will have a firmer, healthier peridental membrane.

It is also necessary for us, as periodontists, to help the pedodontists to prevent periodontoclasia which we do sometimes find in the mouth of the child.

The treatment of gingivitis in the child's mouth should be no different from that in the adult's mouth. There is no condition in the tissues of the child's mouth which is any different from that in the adult's mouth. We can accomplish these treatments if we understand child psychology.

Fifty children between the ages of two and four years were selected at random from our Day Nursery for an experiment. These children were bathed, dressed and fed, and had their own toothbrushes as did the other children in the Institution. These children were seen, some once a month, some twice a month, de-

pending on what I judged was their susceptibility to caries and gingivitis. They were carefully examined, and a prophylaxis was given at each visit. At the end of two years there were no cavities, no stains, and the gingivae were in a healthy condition.

Many times we see a child at two and one-half to three years old who has no separation between the deciduous teeth, no growth of the jaws to make room for the larger permanent teeth. This may be due to mal-nutrition, to endocrine disturbance, or to disuse, more often the latter. The mother, being ignorant as to the function of the teeth, is still giving the child the same soft food it was given at six months. If this condition exists, we cannot expect to find a beginning separation. To correct this, food that will necessitate chewing must be given.

A rubber tongue is used by a prescribed set of exercises to expand the jaws until they are made ready for the permanent teeth. This rubber tongue, six inches long by one and one-half inches wide, is made of red inner tube. In eight to twelve months, a gratifying result is seen. In preventing mal-occlusion we are preventing a gingivitis which will if not corrected in the small child, result in a serious periodontoclasia.

Inflammatory conditions in the gingiva of the child represent a series of oral anomalies which, in some instances, have their origin outside the mouth and in turn, affect bodily health. The classes may be divided into three groups. First, those which are purely local; second, those caused by systemic conditions; and third, Vincent's infection. In the first group, we find the lesions caused by lack of hygiene and by mal-occlusion. By seeing the child early and establishing hygiene by proper use of a well-fitted toothbrush, this condition may be prevented.

Our records have proven that, beginning at the second year, the systematic use of the toothbrush on both teeth and gingiva will prevent a gingivitis. The records also show that correcting a mal-occlusion, when it is the cause, will cure a gingivitis. I have never seen, in group one, gingivitis in the small child which has not been improved and finally cleared up entirely by the co-operation of the patient with the home care and proper use of the toothbrush.

Vincent's infection in the child has its origin in the flap of an erupting tooth. In the small child, as a rule, it is at the time of the eruption of the second deciduous molar. Unlike the adult, when this condition is corrected and the tooth erupted, there has been, in my practice, no recurrence. In the older child the origin is often around the partially erupted first or second permanent molars.

One specializing in dentistry for children can observe all lesions of periodontoclasia more quickly than the pediatrician. In the child we have a beginning gingivitis. It is characterized by a slow and progressive destruction of the alveolar process, suppuration and hypertrophy, gum recession, and loosening of the teeth. We realize the result in adult life. It is one of the most common

foci of infection found in the body. A gingivitis in the small child can be easily corrected once a diagnosis is made as to which group it belongs.

The two greatest things in dentistry should be prevention and diagnosis. It is in the small child that we can stimulate co-operation and form habits that will follow him through life. I have always felt that if my clinic and institutional children could co-operate and prevent periodontal lesions the, supposedly, more intelligent children in my private practice could do the same.

The child should be taught that periodic visits to the dentist are essential. It will be easy, after examining the child, to determine the susceptibility and immunity to caries and periodontal irregularities, and have his visits timed accordingly. I do not think anyone is so immune as to go over three months without an examination. Many I have seen once a week until I had their co-operation.

Preventive work in the mouths of children is considered by many to be chiefly in the realm of prevention of caries and does not pertain to the oral health of the child, or particularly to periodontal conditions. There is, in my opinion, as much periodontal disease as caries in the mouth of the child. In the examination of large groups of children in institutions and clinics, gingivitis and caries are found. The occurrence of gingivitis and caries is simultaneous. Again, we may find gingivitis where there is no caries. We may find gingivitis in the posterior part of the mouth where there are proximal cavities, and where food impaction is allowed to occur against the gingivae. In these cases we find the child using the opposite side for mastication. This habit has sometimes continued for years and serious periodontoclasia has resulted. If the gingivitis is on both sides of the mouth, we will find a pasty, anemic child with lack of resistance to all diseases incident to childhood. Many times in this localized gingivitis, we find bacterial colonies which contain everything from a "yeast cell to a streptococcus." This was the report sent me by a pathologist after making an examination of his own child who had a gingivitis. There is absolutely no function in these cases.

Again we find in the child's mouth a gingivitis caused by the loosening of the deciduous teeth. While the exfoliation of the deciduous teeth is a physiological process, it is often accompanied by conditions which are pathological. Not only the gingiva over the erupting tooth but the gingivae of the teeth, mesial and distal, are involved. Due to the mobility of the tooth, the biting stress of the opposing teeth often cause a painful irritation of the gingivae.

Malposed teeth cause a gingivitis but when these teeth are brought into proper alignment the gingivitis is eliminated.

Salivary calculus is rarely found in the child's mouth due to the fact that, in the intake of food, there is an equal assimilation and elimination. This is due to the strenuous exercise of the child. Only when the child is not allowed to exercise do we find calculus, and then the child lacks resistance and there is no tone to the gingival tissue.

In all cases of gingivitis we find remarkable recovery in the establishment of mouth cleanliness, restoration of function, and stimulation of the circulation by proper massage with a toothbrush.

It is rare to see a generalized gingivitis in the child due to local causes. The only cases I have before me now are from systemic origin, or from an unbroken habit of using one side of the mouth for mastication for several years. It is not difficult with the co-operation of the small child to eliminate gingivitis. If this condition is not corrected in the child, how difficult will be the treatment of periodontoclasia in the adult.

The old adage "large trees from small acorns grow" is applicable to the training of the small child in mouth hygiene to prevent the horrors of a mouth with periodontoclasia. The average small child who is interested in the services rendered him is far more appreciative than the adult, and is more co-operative. I feel that, if in institutional service periodontoclasia can be prevented, surely, it can be done in general practice by each one of us who is interested in and loves the child.

Let us be exacting in our diagnosis and treatment, and prevent periodontoclasia by obtaining the hearty co-operation of the child in his mouth hygiene for true is the saying, "Train the child in the way he should go and, when he is old, he will not depart from it."

My earnest plea to you is that we stress prevention in all that we do in dentistry; especially to the younger men and women dentists who are starting down life's paths armed with forceps, lancets, hypodermics and elevators, and who only see and believe that there can be but one end result in dentistry and that result, edentulous patients. They should be instructed in their pre-dental courses in the art of prevention.

In closing I have one recommendation to offer—that we lay more stress on the *prevention* of periodontoclasia and dental caries.

1203 *Maison Blanche.*

NOTICE TO MEMBERS

The Editor has received requests from several dental libraries for the January, 1930 issue of the Journal of Periodontology to complete their files of this publication. The co-operation of members in supplying this need will be greatly appreciated.

Address communications to 555 W. Maple Ave., Birmingham, Michigan.

Incidents of Office Practice

Method of Taking Case History

By Frederick A. Bricker, D. D. S., F. A. C. D.
Los Angeles, California

The consideration of constitutional factors in the treatment of periodontoclasia is of vital importance and I place greater stress on it than upon the local factors.

The case which presents a picture of every condition conducive to periodontoclasia, trauma, filth, etc., and has no resorption or inflammation probably has the general resistance up to par. In contrast there is the case which should and does not respond to treatment because the general resistance may have been lowered by anemia, diabetes, glandular disturbance, or other systemic involvements. In each of these cases we must consider the influence of the systemic factor.

HISTORY

In my experience it has been most valuable in diagnosis and treatment to obtain and record information concerning the following systemic influences:

1. *Chief complaint.*

What symptoms caused the patient to seek relief?

2. *Childhood history*

Diseases of childhood, (measles, mumps, scarlet fever, etc. which may explain the condition of the enamel on some of the teeth). Also record if patient had a healthy career during childhood. Where was patient born and where lived, on farm or in city? Hereditary history. Family tendency toward periodontoclasia. Diet during childhood.

3. *Adult history*

Record any severe illness, surgical operations, asthma, constipation, frequent colds, bowel rate, gastro-intestinal conditions, diabetes, anemia, endocrine gland disturbances, menstruation, abnormalities, menopause, nephritis, nervous breakdown, gastrectomy, gastritis, condition of tonsils. Any one of these abnormalities may be reflected in the mouth.

Record information regarding exercise, shallow breathing, rest, nervousness, irritability, despondency, easily fatigued, exhaustion, alcoholics, smoking, appetite, sleep.

Nutritional habits. Information on diet, amount of milk, vegetables, salads, fruits, grains, meat, water.

Type of patient: Neurogenic, emotional, tempermental, worrying, despondent, phlegmatic. Note condition of skin, flabby or firm, dry or oily. Get *patient's statement* of what operations or illness he has had, jot it down, and transfer to regular history chart, instead of enumerating all the systemic disorders found on a diagnostic chart. It *simplifies* taking this history.

Laboratory tests. Complete blood picture, urinalysis, and if history suggests it, basal metabolism and blood sugar tests.

Copy of one week's diet. Instruct the patient how to keep this record for you. Everything he eats and drinks (tea, coffee, stimulants, drugs,) must be included.

A good full mouth x-ray to study the bone structure especially the trabeculae is essential. Note resorption of alveolar bone.

Examination of the mouth. Note mobility of the teeth, color, form, density, line of attachment of the soft tissues, hygiene, trauma, etc.

With this information at hand, a judicious, careful study of the history very often reveals a causative factor that may spell success or failure.

A great many of our failures to obtain successful and lasting results can be attributed to the tendency of treating the case as a local disturbance rather than a general. In making a diagnosis and prognosis it is important to consider all systemic factors which bear any relationship to the periodontal condition, as well as all of the local factors.

A Stubborn Case of Vincent's Infection

By John H. MacKay, D. D. S.
Oakland, California

I have in mind a case of Vincent's Infection which I believe will be of interest to this group. The patient, a young man in his early thirties, stated that the original infection occurred about ten years previous. The unusual thing about it was the fact that it has recurred every six weeks since then, and that it had started in the same area each time. As you all know, most cases of Vincent's infection are potential cases of paradentosis, and that it is not enough to treat the acute symptoms only but thorough subgingival curettage is necessary, and last but not least, the occlusion must be checked and any undue stress relieved.

In examining the anterior bite of the above case, I found that in this position the only one of the lower anterior teeth in occlusion was the left lateral incisor. It was from this area that the infection originated and seemed to spread each time. The occlusal stress was taken off of the lower left lateral incisor and placed upon the six anteriors by grinding. I am happy to state that the patient has had no recurrence of the infection since. He was the most skeptical patient I had ever had until the sixth week had come and gone.

In conclusion, I would emphasize that cases of Vincent's infection be looked upon as potential paradentosis and that they be treated as such. If this is done, then the fact that a patient acquires Vincent's infection would be a blessing in disguise for he would then receive preventive treatment for paradentosis, which if not given would probably lead to the early loss of the teeth as most cases are well established before they are discovered or diagnosed.

Medical Center Bldg.

The Treatment of Extensive Periodontoclasia of the Lower Anterior Teeth

By R. Dean Creamer, D. D. S.
Phoenix, Arizona

If the case history shows no organic disease, and the blood picture good, I consider treating these extensive cases of periodontoclasia.

Thorough clinical and x-ray examinations are made to determine the position and density of the bone structure, and the depth of the pockets around the teeth which is recorded on charts for the purpose.

Since we know that arterial blood is necessary for the repair of tissue, it is my purpose to teach the patient how to massage the gum tissue in the interproximal areas with the toothbrush. I always demonstrate the use of the toothbrush in the patient's mouth, after which he is asked to show me if he can handle it correctly. Sometimes it takes two or three sessions of instructions before the patient becomes efficient in the personal care of his mouth.

Subgingival curettage is the preferred method of treatment in my hands. When removing calcareous deposits from deep pockets I use local anesthesia. Instrumentation should be accomplished with as little irritation as possible but I believe that patients prefer anesthesia, and it makes operating more convenient.

My instruments are of the plane type except one, which is used to take off the heavy deposits from above the gum line. Where there is extensive destruction about the lower incisors I usually scale three teeth at one sitting, trying to remove all deposit and plane the root surface smooth at that time. At a later appointment my assistant holds a transillumination light on the lingual while I dry with air and examine the teeth for any remaining deposits on the labial and proximal surfaces. This procedure is also carried out using the light on the labial surface. With the light in position and while the teeth are dry, I remove the remaining deposits as carefully as possible to prevent bleeding. If there is enough bleeding to prevent accurate scaling, these

areas are checked again at another sitting. McCall's epithelial solvent (sodium sulphide) is used in all pockets after I am sure that the teeth have been perfectly scaled. The area is isolated with cotton rolls and the sodium sulphide solution is introduced into the pockets on small wisps of cotton or silk tape, packed in with a blunt probe. These are left for five minutes, and after removing them the tissue is massaged with a pinching action of thumb and finger. This procedure may be repeated at a future sitting if it is considered necessary.

Traumatic occlusion is one of the major causes of periodontal destruction. Impacted third molars are frequently responsible for trauma on the anterior teeth. Due to the sustained pressure of the third molars in their effort to erupt, all of the teeth may be forced forward, causing the lower incisors to strike very hard in centric occlusion, and if the pressure is allowed to continue, lapping of the centrals, laterals, and cuspids will be noticed. I recommend the extraction of all third molar impactions to prevent further movement of teeth in the arch.

In correcting the occlusion the incisal edges are ground so that a thin piece of paper cannot be held between the teeth in centric occlusion. The anterior teeth are ground, if possible, so that all of them contact in the incisal bite. When any anterior tooth is movable an effort is made to take it out of occlusion entirely. The patient is asked to bite laterally in order to check traumatic occlusion in the excursions of the bite. Two or three weeks after the occlusal correction is finished, the patient is instructed to return to have checked the contact of all the teeth in centric, incisal, and lateral occlusions. Due to the contraction of the tissue in the healing process the teeth usually tend to move to their original positions, making further occlusal correction necessary.

After occlusal correction and subgingival curettage are completed, the teeth that are movable are stabilized by some appliance. In cases where loose teeth are lingual to others that are stable, a retainer of vulcanite rubber is made to fit the lingual surfaces of the teeth extending down to the tissue attachment and back to the distal of the second molars with a stop wire to hook on to the occluso-lingual groove of the first molars—a retainer similar to those used in retaining the lower teeth in orthodontic cases. In cases where the loose tooth or teeth, are in line with or labial to, stable approximating teeth, I use an orthodontic band on the teeth on either side of the loose ones, and solder a small round wire from one to the other on the labial and lingual, adapting the wire to conform to these surfaces. In this way the teeth that are loose are held immovable from one to two years, giving time for rebuilding of tissue and bone.

I have cases, treated six and one-half years ago, where the alveolar destruction extended down to the apical third, which are healthy at this time. The tissues show considerable recession, but the teeth have become firm or have very slight movement with pressure.

In conclusion I consider that treatment of these extensive cases of periodontoclasia is warranted by the results obtained in my practice over a period of seven years. No doubt you have all had similar results.

820 *Professional Bldg.*

Indication and Contraindication for Subgingival Curettage in the Treatment of Paradentosis*

By Dickson G. Bell, D. D. S.
San Francisco, California

There is a great deal of controversy today regarding the different types of treatment for paradentosis—this is particularly true with the so-called conservative method or subgingival curettage. Perhaps the writer's conception of the indications for treatment will be entirely different from that of the men following the surgical method, even different from those who use subgingival curettage exclusively or only in certain types of cases.

The question arises whether subgingival curettage will accomplish the desired results in every case. The following observations are based on clinical experience and the fact that this particular method of treatment has been used by the writer for many years. The proper application of subgingival curettage will eliminate the infection, reduce hypertrophied tissue, prevent additional resorption of the alveolar margin, and in most cases establish an attachment or organic union of the separated soft tissues with the cementum of the tooth, thereby closing the pocket; in brief, accomplish the main general objective in the treatment of paradentosis, i.e., to eliminate the inflammatory process and restore the normal physiology of the paradentium. I fully realize that there are other methods which probably have their place, but frequently they do not produce the desired results. Successful treatment of paradentosis by any method is always predicated on the fact that patients follow a satisfactory regime of home care and receive correct periodic prophylaxis.

Every treatment in medicine as well as dentistry must have a definite indication. In the treatment of any lesion of the organism, we cannot assume that surgical procedures are the only methods of choice. We know today that gastric ulcers, duodenal ulcers, kidney diseases and others may be arrested or cured in many ways besides surgery. However, each method has its definite indication, which in itself is determined by factors favorable to it.

In paradentosis, we have many factors which render the prognosis of the treatment unfavorable. This applies to subgingival curettage as well as to other methods.

One of the first factors to be considered as being somewhat unfavorable is exposed dentine on the root surface. This condition, due to the loss of the cementum, is found mostly in localized areas and easily identified by a sensitiveness to mechanical, thermal or chemical irritation. It seems to be an unfavorable factor for several reasons. In the cervical region, when these exposed root surfaces are sensitive, the patient is reluctant to brush these areas properly, with the result that a chronic gingivitis is usually present. In other cases, we find the gingival margin split root-wise for 2 or 3 mm, and if the underlying root surface is exposed dentine, it is often difficult to obtain a satisfactory healing of the tissue. I do not think any scientific evidence has been presented to explain this clinical observation; however, these lesions will quickly respond to treatment when the surface is not sensitive. Therefore, it seems logical to assume that when a layer of cementum is present, that fixation of the sub-epithelial connective tissue to the surface of the root will occur more readily.

There is also the problem of exposed dentine in the subgingival region, which, fortunately for those of us who practice subgingival curettage, is only occasionally found in an individual case or on a few isolated teeth. There are two reasons for the destruction of cementum in this particular area, (1) chemical, caused by acids formed in the pockets as a result of bacterial fermentation, usually found close to the gingival margin and (2) mechanical, in cases that have been treated by an over zealous fellow practitioner who unknowingly cut through the thin layer of cementum and exposed the dentine.

One of our main objectives in cases of paradentosis, as far as local anatomical conditions are concerned, is to close the pockets,

*Read before the American Academy of Periodontology. San Francisco, California, July 9, 1936.

because we consider them as being somewhat of an irritating factor in the health of the paradentium. However, in cases where this ideal condition is not obtained, there is a close apposition or contraction of the tissue over the region of the exposed dentine. Clinical observation of such cases has shown that over a period of time—when the patient returns for subsequent prophylaxis—many of these exposed areas are no longer sensitive and the tissues have apparently assumed a normal condition.

A second unfavorable factor is the involvement of the bifurcations of molars, and this is undoubtedly one of the most frequent problems that confronts the paradentist in his prognosis. The paradentium adjacent to these bifurcations appears to be quite vulnerable to pocket formation, and it is very difficult to secure favorable results. Apparently there is the mechanical factor of a groove or narrow concave surface which is very susceptible to irritation and infection. This same anatomical form will in reverse order affect the character of healing. It has been observed that rapid healing depends to a large degree on a close approximation of the soft tissues to the cementum of the tooth. Apparently, in these bifurcated areas, the tissues are not always sufficiently close to the cementum for this healing to occur. As the inflammatory process is reduced, the tension of the gingival margin may prevent the biologic tendency of the regenerated tissue to follow these concavities. It also seems more difficult to obtain attachment of the tissues to a concave surface than it is to a convex one—for instance, the mesial surface of an upper first bicuspid.

Even though the involvement of a bifurcation is a decidedly unfavorable factor, the writer does not believe that all such involved teeth should be extracted. There are many situations in which one's judgment must determine the procedure to be followed; for example, a bridge abutment with moderately deep pockets, an upper molar with shallow pockets or even a moderately deep pocket on one surface, lower molars with pockets of medium depth, etc. In other words, some particular circumstance may exist which makes it necessary to tolerate such teeth, and therefore treatment is indicated on this basis. Such teeth, when treated by the subgingival curettage method have responded very well.

Clinical experience has shown on subsequent examination that many of these pockets do not appear to be as deep as they were originally. It must be assumed therefore, that some healing takes place at the bottom of the pocket. An improved tonicity of the tissues is obtained, resulting in a close adaptation or apposition of these tissues around the root surface. Occasionally we find a groove on the lingual surface of upper lateral incisors, and this same premise may be applied to treatment of a paradental pocket on that surface.

The third unfavorable factor may need to be considered when teeth have lost over two-thirds of their alveolar support. They are usually a poor risk, not so much from the standpoint of pocket formation as from the fact that such teeth frequently become increasingly loose and show signs of migration. Under stress of mastication this mobility becomes a source of trauma, thus resulting in an irritation of the tissues.

There are some exceptions to this general rule, of course, particularly in the anterior region where it may be advisable to treat such teeth. Treatment of deep pockets on the lower incisors is frequently indicated and they are quite favorable, especially so, if they are in occlusion with an upper denture. However, in cases where such teeth are treated, they should have temporary splints to stabilize them until such time as the tissues have become firm and attached to the root surfaces, thereby giving them a natural support. Also, the occlusion should be carefully balanced and any anatomical or mechanical defects corrected by operative measures.

There is another condition which must be taken into consideration in these particular cases, namely, bone density. Our improved roentgenographic technic has shown that there is quite a difference in the structure of alveolar bone. In some patients, it appears dense and compact, in others, due to vitamin deficiencies or endocrine dysfunction, it is more or less radiolucent. If the latter, the osteoporotic type is believed to exist, then subgingival curettage must be supplemented with simultaneous treatment of the systemic disturbances.

Another factor which must be considered in the prognosis of every case of paradentosis is occlusal trauma. The different conditions which cause abnormal stress need not be enumerated as they are well known to all of you. The result of occlusal overstress depends to a large extent on the local reaction of the tissues to an increased functional requirement. In other words, the paradentium of one individual reacts with a breaking down of the osseous structure, while in another case it will roentgenographically show a building up. In my opinion, the theory of Stillman and McCall that occlusal overstress is one of the outstanding causes of paradentosis, applies only to certain individuals—those who have a "readiness" to paradentosis. In probably the majority of cases under our observation, we find that the mobility, migration (except as a result of extraction), and elongation of teeth is the result of primary atrophic or inflammatory conditions of the paradentium.

In any case, occlusal trauma is an unfavorable factor which must be corrected. This should be done at two different periods: (1) before treatment of the pockets, when only sufficient grinding is necessary to relieve excessive movement of the affected teeth, and (2) after completion of local treatment, when the correction is carefully finished, so that all teeth will be in as favorable occlusion as possible. The reason for this procedure is based on the previous statement, i.e., retrograde changes in the paradentium are a primary factor in causing occlusal trauma. Clinical experience verifies it, because when the tissues are restored to a state of physiologic health, the teeth frequently return to their original positions.

There is also the problem of pulpless teeth. Clinical observation has indicated that when detachment of the paradental tissues occurs on a pulpless tooth, it is an unfavorable factor for prognosis. The cementum of these teeth does not seem to be capable of producing an organic connection with the detached epithelium—perhaps because the metabolic exchange between the pulp canal and the root surface through the dentine and cementum has ceased to exist. Why the surface of the tooth acts differently after the pulp tissue has been removed than it does in those cases where we are dealing with a vital tooth has never been established—however, it is general clinical experience.

Our knowledge of the infectious nature of pulpless teeth and their influence on systemic conditions, determines to a large degree the procedure to be followed. The fact that many of these teeth show roentgenographic evidence of apical infection, necessitating extraction, will in such cases eliminate the problem of paradental treatment. However, there may be certain exceptional cases in which it is necessary to retain pulpless teeth, provided of course, the physical condition of the patient is such that he may tolerate them. Under these circumstances, treatment of paradental pockets of moderate depth is indicated, and a decided improvement in the health of the tissues is obtained by subgingival curettage.

It seems to be very well established that not all types of gingivitis are caused by local exogenous factors, and the types which cause us the most trouble are those occurring on the basis of an abnormal systemic background.

One type. of gingivitis frequently observed is that associated with pregnancy. Ziskin *et al*, report that an examination of the mouths of 416 pregnant women showed 158 or 37% with some form of "pregnancy gingivitis." It is interesting to note that Stoloff reports a syndrome of changes in the gingival tissues of women just before menstruation, in acute functional gynecological disturbances other than pregnancy, during and after the menopause, etc. The tissue changes may be of a mild character, such as redness with a tendency to bleeding when traumatized by the tooth-brush, food, etc., or a more severe form in which there is a hypertrophic gingivitis—also, what appears to be an overgrown septal gingiva or small pregnancy tumors. If the local irritants are kept under control by prophylactic measures, the gingivitis usually disappears at the end of the pregnancy period.

There is another type of gingivitis which we frequently observe during the adolescent period. The fact that it occurs at puberty leads to the assumption that the causative

factor is the appearance of the genital hormone. (Roy), This gingivitis may exist for a year or so and then disappear, presumably when the glandular condition has adjusted itself.

In both of these types of gingivitis, local irritations seem to be a secondary factor, as mild forms have been observed in patients who received periodic prophylactic treatments. Since the gingival tissues are edematous in these cases, they bleed readily and are painful to brushing, consequently the patient is somewhat reluctant to thoroughly brush the teeth, with the result that there is accumulation of bacterial plaques and food debris. Therefore, the value of careful mouth hygiene during these periods should be impressed on the patients.

Deposits of calcium salts may form on the underlying tooth surface as a result of the prolonged vascular disturbances, and of course further aggravate the condition. Therefore, subgingival curettage is indicated in the treatment of these cases of gingivitis, to remove this irritation, thereby reducing the inflammation to the minimum.

The possible relationship of nutritional deficiencies and systemic disturbances to paradental lesions has already been discussed. Although at the present time, these cannot be considered as definite etiologic factors in the production of the disease, yet experimental and clinical investigations seem to indicate that we should look more than ever into the question of the physical condition of the patient, if we wish to approach this problem of paradental treatment in the most intelligent manner. Therefore, in these particular cases, local treatment may be considered as a procedure of removing only the clinical symptoms, because up to the present time we have done very little to simultaneously treat the systemic etiologic factors of the disease. Perhaps this may account for a certain percentage of our failures.

The several factors which I have enumerated are some of the stumbling blocks in the treatment of paradentosis. There are, of course, many other unfavorable conditions, such as ulcerative gingivitis or paradentitis, anomaly in position of the teeth, lack of contour in restorations, loss of contact points, personal habits of the patient, long-standing chronic cases in which the general practitioner has repeatedly attempted to treat the paradental lesions, etc.

It is theoretically possible that epithelial and connective tissue can re-attach itself to the surface of dentine. However, from a practical standpoint, it seems as if our clinical experience speaks against this possibility. It is frequently found that in so-called "over-scaled" cases, i.e., in cases where the cementum has been removed by scaling and also the superficial layers of dentine, that the pocket does not close up as satisfactorily as in cases where the cementum surface has remained intact. The same observation applies to pulpless teeth, It is for the future to solve these problems by experimental means, and only histologic evidence with accurate case reports can be accepted as final proof.

The Price of Professional Progress

TO KEEP abreast of the progress being made in the practice of dentistry is no longer an easy task. So rapid has that progress been in the last twenty-five years, that no one can hope to keep pace with it who is not willing to face facts and pay the price. The time is past, if indeed it ever existed, in which the dentist can confine himself to his office activities and expect to discharge his obligation to society.

Commencement day, for the graduate in dentistry, is but the beginning of professional life. What the end will be—progress or stagnation—will depend upon the individual and the effort which he makes to keep step with an advancing profession.

There are several ways in which this may be done. One is through observation

and experience. All learn more or less in this way. It is a step, and an important one, in the right direction. But it is only a step. A full measure of success is impossible to the dentist who limits himself to experience acquired in his office. Without realizing it, he gets into a rut; his horizon is limited by the walls of his office.

Another way is through study and reflection. This is a matter of the utmost importance. Every dentist should begin early in his professional career to form a good working library. He should begin by subscribing to half a dozen or more good journals, including at least one medical journal. With these at hand he should form the habit of systematic study in order to keep in close touch with his profession. At the end of each year, these journals should be bound and made available for future reference. This cannot be urged too strongly. Their value is beyond price. The writer speaks from experience. He has in his library complete bound volumes of five different publications, including the Dental Cosmos, the Journal of the American Dental Association and the Journal of Dental Research. They have been to him a never failing source of help and inspiration.

To these should be added as many current books each year as one can afford to buy and find time to read including two or three good dictionaries and at least one medical dictionary. Never were there so many good books being written and published as at present. This is one of the signs of progress for they represent the advanced thought of the profession. No one can achieve success in his profession who neglects the habit of regular study.

A third and very important way in which progress is made is through attendance at dental meetings. Experience and study are necessary but they are not enough for a well rounded professional career. One needs the inspiration and help which comes from association and cooperation with ones colleagues. Every member of the profession should, therefore, affiliate himself on graduation with organized dentistry and take some part in its activities. In this way one forms a wide acquaintance among his co-workers and comes face to face with them in the discussion of their common problems. Observation and study should equip one to take some part in the programs that are being presented. Not only does one in this way, make his contribution to his profession, but his own mind is enriched and broadened. The writer of a paper is always the chief beneficiary. But whether or not one takes part in the program, he should form the habit of regular attendance. The writer has had some experience in attending dental meetings and out of that experience he would say that he has found it to be the greatest educational influence in his professional life. Not only has he learned much that has been of great practical help to him in daily practice, he has, in addition, received inspiration and encouragement such as he could have found in no other way. Then too, there are the friendships one makes. Who can estimate their value or measure their influence? The writer cannot imagine what his pro-

fessional life would have been without the help and encouragement of friends, many of whom he would never have known had he not formed the habit of regular attendance at dental meetings. The friendships made in this way, plus the knowledge that has made possible a better service, have repaid a hundred fold any sacrifice which he has ever made. This will always be found to be so, for the price of professional progress is never high when compared with the reward which comes to those who are willing to pay the price. And the price which all must pay who would succeed is observation, study and coöperation.

A. H. M.

Committee Reports

Report of Committee on Ethics*

Your Committee on Ethics hereby reports that the only matter requiring their attention during the year was the instructions from the Council to send a letter to each member of the Academy calling attention to Section XI of the Code of Ethics, on Journalism, and requesting their cooperation in compliance therewith.

The letter was sent as instructed. A copy of the letter follows.

Respectfully submitted,

W. O. Talbot, Chairman,

Dear Member:

As chairman of the Ethics Committee of the American Academy of Periodontology and under instruction from the Council, may I call your attention to Section XI of the Code of Ethics which reads as follows:

> The members of the Academy are urged to give their support to every effort that is being made to promote professional journalism. They are likewise urged not to accept positions on editorial boards or to lend their influence to trade journalism by the preparation of articles for publication in such journals.

Believing that this section of our Code has been overlooked by some members in accepting membership on the editorial board of such publications or by contributing to their pages, the Academy expresses the hope that they will after further consideration feel as it does, that this is hardly in keeping with this section of our Code.

It is the Academy's belief as expressed in its Code of Ethics, that we can never hope to purge our literature from the taint of commercialism as long as those whom we have learned to look up to in the profession, continue their support of that type of journalism which has the disapproval of organized dentistry.

It asks the cooperation of every member in its efforts to hold high the standards of professional ethics.

Sincerely yours,

W. O. TALBOT, *Chairman*

Report of the Committee on Subscriptions for the Journal of Periodontology*

An effort was made to contact the Academies of both California and the Southern States. The roster of the California Academy was received and subscription cards were sent to the secretary, to be presented at their regular monthly meeting—with the result that some subscriptions to the Journal were added.

The most active and successful member in obtaining subscriptions, is not a member of the Committee. At two different meetings, in one week, Dr. Merritt secured the names of forty-nine who expressed their desire to subscribe. Of this number twenty-three are paid up and probably all will soon be subscribers.

* Read before the American Academy of Periodontology, July 9, 1936, San Francisco, California.

For the reason that the Journal is a contribution of the Academy to Dentistry without the financial assistance of advertisements, it is very desirable that the number of subscriptions be greatly increased.

It is recommended that each member do all he can to secure subscriptions among his fellow-practitioners in his own city—and that we especially concentrate our efforts —and set as our goal for the coming year one or more subscriptions for every University and College library.

Should a college library not be able to subscribe, perhaps some Academy member, living in that city would pay the subscription for the library. This is being done at present by two of our members.

It may be of interest and enlightening to the members to quote part of a letter recently received by our editor from Dr. Thomas F. McBride, University of Pittsburgh.

"My dear Doctor: Today, I received a complimentary copy of the January, 1936, issue of the Journal of Periodontology, and without any delay, want to express my thanks, as well as to congratulate you and the Editorial Board for publishing what is easily the most outstanding journal devoted to a specialty in the entire profession.

The comments of the Commission on Journalism in 1932, concerning your journal, still hold. It would be well if not only other specialties followed your lead, but that some of the other publications do likewise.

The cover, general appearance, type-face, arrangement—the entire format is conceived with dignity and professionalism. Its make-up *wants* you to read it. And there is no let-down after doing so. We have your journal on file in our library, and I have been familiar with its appearance and contents in general, but today was the first time I had ever read it through. Believe me, it shan't be the last time.

The articles are indeed instructive. They have a certain flavor of scientific accuracy about them, and soundness of thought that is found in only too few of our periodicals. Especially did I enjoy the article by Dr. Merritt concerning the Curriculum Survey Report and Periodontology."

The Subscription Committee greatly appreciates the efforts of all members of the Academy who have obtained subscriptions.

Respectfully submitted:

MILDRED DICKERSON
STELLA RISSER
GRACE KEITH PULLIAM, *Chairman*

July 9, 1936,
San Francisco, California

The President of the American Academy of Periodontology 1936-37 sends Greetings and a cordial invitation to "you all" to attend the Twenty-fourth Annual Meeting July 8, 9 and 10 at Atlantic City. The chief inducements are 9 essayists, 6 scientific reports in addition, 9 clinicians, 20 leaders and 2 directors for the Round Table Discussion Luncheon, and the Banquet, always a delightful event, with DR. WILLIAM J. GIES as Speaker, MRS. CARLOS H. SCHOTT as Soloist, and MRS. JOHN H. MACKAY as Reader. Can you afford to miss this record making Academy of Periodontology program?

BOOK REVIEWS

Black Ivory. By Edward Samson, L. D. S., F. C. S., England. 179 pp. 1936.

Again, from the literary pen of the author of *Common Sense Dentistry, Dental Doses,* and other gems comes *Black Ivory.*

This work, an indictment against civilization for its *laissez faire* attitude toward dental caries and pyorrhea is written in a fine philosophical manner. In fact, the treatment of the life on the island of Tristan Da Cunha somehow reminds one of the allegorical descriptions in W. H. Hudson's *Green Mansions.*

The chapter "White Ivory" describes vividly the existent dental Utopia where on the little island seventeen hundred miles from the Cape of Good Hope in the South Atlantic there is a colony of people completely devoid of dental disease or in fact any sickness. People there die either from old age or accidents—they have not suffered disease for over a century.

Black Ivory will go down in the annals of history as a monumental accusation against all economic systems of the world for their lack of cognizance of the greatest scourge ever to affect humanity and which strikes prince and pauper alike even today as it did to Sir Arthur Keith's Rhodesian man. Dental decay or Black Ivory, the author believes, is the underlying cause of most body and mental disorders. "Yet," he writes, "a man walks about half his life with a foul mouth, ultimately dying from some disease it has caused; nobody knows except the doctor—he will write on the death certificate something as remote from dental sepsis as heart failure."

Dr. Samson in his reveries on faults in economic systems takes a poke at our "Prohibition" and all the chaos it caused for a while. However he observes that when ninety per cent of a population suffers from a scourge almost as old as man, governments neglect it and concern themselves with personal behavior problems like drunkenness, child marriages (in India), or companionate marriage.

We highly recommend this fascinating book to every dentist in America who has at heart the welfare of posterity. It not only summons our righteous indignation concerning the vast neglect of government in so important a matter, but Dr. Samson in his inimitable British style gives many valuable suggestions as to how humanity might be benefited by government research; of course under expert professional guidance.

Using "customary American enthusiasm" which the author entertainingly suggests ought to improve our race we say by all means read *Black Ivory.*

—*Copy, from Items of Interest, Nov., 1936.*

A Dictionary of Dental Science and Art Comprising the Words and Phrases Proper to Dental Literature, with Their Pronunciation and Derivation. By William B. Dunning, D. D. S., F. A. C. D., and S. Ellsworth Davenport, Jr., D. M. D., F. A. C. D. 635 pp. 79 illustrations. 1936.

"The compilation of a dictionary is by no means an easy task. Dunning and Davenport have unquestionably accomplished an arduous and exceedingly tortuous undertaking for which they are to be commended. Their painstaking efforts have culminated in the publication of *A Dictionary of Dental Science and Art,* which they present to the dental profession as a comprehensive dictionary of dental and oral terminology and of the collateral but strictly related science. Their object "has been to produce a book of practical value; one that includes the best of the past with that of today."

"This book is the fourth of its kind in English Dental Bibliography. The previously published dictionaries are those of Harris, Ottofy, and Anthony, which appeared in the respective order mentioned."

"A feature of this dictionary is the inclusion of thirty-four portraits of outstanding men in the dental and medical professions, with their full names, birth and death dates, and countries of origin. This will be of great value to dental practitioners interested in historical data. While the portraits of such celebrated characters as Fauchard, Hunter, Miller, Black, Brophy, Dunning, Darby, Rhein and others appear,

the portraits of Cryer and Kirk are omitted. It is also surprising that the names of such eminent men as Goethe, Lemonnier, Carl Ferdinand von Grefe, Philibert Joseph Roux, Dieffenbach, Bernhard Rudolf Konrad von Langenbeck, Billroth, Julius Wolff, Blandin, Bolk, Rose, Kolliker, Trelat, Sedillot, Warren, Evans, White, Hopewell-Smith and many others—men with outstanding contributions in oral surgery and dental science and art—are not mentioned at all.

"A large number of proprietary preparations have been included such as Lavoris, Pepsodent, Mercitan, etc., yet such preparations as percaine, nupercaine, cobefrin, mapharsen, dentocol, elastic impression, resovin, luxene, etc., are omitted. Likewise many articulators in use have been cited, with the exception of those of Wadsworth, Hanau, Needles and Lentz.

"This dictionary contains a number of useful tables covering the various anatomical entities of the head and neck (bones, muscles, arteries, veins and nerves), the elements, microörganisms of the mouth, and weights and measures. The table covering microörganisms of the mouth, however, does not include the Bacillus acidophilus odontolyticus and the Streptococcus mutans, which are found in dental caries."

"Adequate space is given eighty illustrations, some of which are in color."

—Extract from Review by E. Shirazy, D. Cosmos, 1936.

E. Shirazy's review includes additional interesting critical paragraphs calling attention to errors and omissions, but concludes with the following statement, "A Dictionary of Dental Science and Art is valuable as a ready reference handbook, and no dental library is complete without the inclusion of this book among its archives. The book is unquestionably a valuable addition to our English dental bibliography."

—C. R.

"Studies in the Aetiology of Dental Caries." By Graham Macphee, M. A., M. D., Ch. B., L. D. S., Honorary consulting Dental Surgeon, Liverpool Stanley Hospital; Honorary Assistant Surgeon, Liverpool Dental Hospital; Honorary Dental Surgeon in charge, Pathological Laboratory, School of Dental Surgery, University of Liverpool. 203 pp. 1935.

Dr. Macphee's monograph entitled *"Studies in the Aetiology of Dental Caries"* is a unique contribution, being the only complete treatise in English on the subject of caries. This outstanding contribution includes the most important theories advanced to explain the causation of this widespread dental lesion.

The book consists of twelve chapters covering every aspect of dental caries. The first of these chapters deals with the incidence of dental caries, in prehistoric, ancient, medieval, primitive, civilized and modern man. The next chapter answers the question: What is dental caries? The following eight chapters evaluate the various theories advanced concerning the causation of caries. Chapter ten contains the author's observations and criticism of the already advanced theories. Macphee states that while "the actual specific cause" of caries remains a myth, "it is a not unreasonable hypothesis to assume that dental caries, like most other diseases, may be caused by some *contagium vivum*, either a microörganism or a filter-passing virus." This view "of the possibility of a filter-passing virus being the causative agent appears to have been overlooked."

The eleventh chapter of Macphee's book covers the author's orginal investigations and research on this much-dreaded dental caries. Macphee found "a definite precipitin reaction" occurring "between filtered or centrifuged emulsions" of carious dentin and serum from the same or other subjects.

The last chapter sums up Macphee's conclusions. The author is of the opinion that "if further investigations confirm the truth of the hypothesis that some *contagium vivum* is the causal factor, it is not too much to hope that some day dental caries may be controllable by prophylactic measures such as vaccines, just as are smallpox, rabies and yellow fever.

Macphee's excellent monograph is further enhanced by the inclusion of a complete bibliography covering 337 different references, which sum up the writings of the leading contributors on this subject.

Macphee treats his subject in a scholarly fashion. His writing is clear, lucid, to the point and free from dogmatism. The studies he made must have been exacting, demanding considerable labor and research.

"Studies in the Aetiology of Dental Caries" is unquestionably an invaluable monumental contribution which dental practitioners, investigators and students will find comprehensive, interesting and most illuminating. The book is highly commended to the serious consideration of the dental profession, and it should find an estimable place in every dental office and library.

Messrs. John Bale, Sons and Danielson, Ltd., have produced a delightful monograph in keeping with a high standard of British publishing houses. They are commended for making the price of this valuable and highly specialized book within the reach of the most modest pocket.

—*E. Shirazy.*

—Copy (abbreviated) from *Dental Cosmos* —September, 1936.

Dental Infection and Systemic Disease. Second edition revised. By Russell L. Haden, M. A., M. D., 163 pp. 1936.

The question of focal infection in general and of dental focal infection in particular is still of paramount importance to both medical and dental practitioners.

The publication of papers recently in this country, chiefly by English authors, shows that our conception of the importance of dental sepsis and its relationship to systemic disease is fast becoming crystallized. With the passage of the years a much clearer perception of the importance of focal infection in relation to systemic disease has arisen, due almost entirely to the fact that the pathology of dental lesions is more understood. It is now recognized by most teachers that the treatment of dental sepsis does not necessarily mean the ruthless extraction of teeth, and a more rational conception of dental sepsis and its prevention is gradually dawning. Patients and dental practitioners are demanding some measure of proof that septic teeth are the cause of ill-health before extracting them on speculation of benefits which may or may not occur.

The author of this little volume is an ardent disciple of Rosenow, and there is no doubt that his theory of "elective localization" is an honest attempt to assess the importance of dental sepsis in relation to systemic disease. A large part of the present volume is occupied by a description of cases purporting to support Rosenow's contentions. In this country (England) these theories have not been sustained, which invalidates to some extent the conclusions at which the author arrives. There are undoubtedly many statements in the book which can be criticized, but with the general excellence of the contents few will cavil.

The author takes as proof of infection of a tissue the fact that he has cultivated bacteria from it, but has not supported his statement by any histological evidence of any change in the tissue. It is difficult to reconcile the mere presence of bacteria in a tissue as evidence of infection unless that tissue shows some evidence of inflammation. This seems of some importance if there is not radiographic evidence to support the bacteriological findings, though it must be admitted that most dental practitioners think that pulpless teeth, for instance, are infected, even if radiographic evidence of bone absorption is lacking.

Stress is laid by the author on the importance of the oxygen tension as a factor in the growth of bacteria—a point that has always been stressed by Rosenow.

The author has no suggestions to offer as to the reliance that can be placed on serological and blood examination as factors that might help to correlate the dental and systemic lesions.

The chapter on the management of dental infection shows the modern trend, in that the author suggests that the removal of dental sepsis should be undertaken as early as possible. With this we must all agree, and would add that the most satisfactory methods would be those directed towards prevention.

The value of this little volume must hinge to a large extent on the acceptance or otherwise of Rosenow's theory of "elective localization," and at present it must be admitted that research workers in this country have failed to substantiate his findings.

—Copy from the *British Dental Journal*, August 15, 1936.

THE Journal of Periodontology is lovingly dedicated to the memory of Doctor Gillette Hayden. Her selfless devotion and untiring efforts in behalf of periodontia and the American Academy of Periodontology, have served as an inspiration to her close associates which can only be consummated by carrying onward the work for which she spent her life.

THE JOURNAL OF PERIODONTOLOGY

Published twice yearly—January and July—by

The AMERICAN ACADEMY OF PERIODONTOLOGY

Publication and Editorial Office: 555 W. MAPLE AVE., BIRMINGHAM, MICHIGAN

Single copies of this number may be obtained from
Publication Office at $1.00 each
Subscription Price $2.00
All foreign subscriptions to be paid in United States money

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